Occupational Disease and Workers' Compensation in Ontario

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Report prepared for:

Professor Paul C. Weiler in his study of
Workers' Compensation in Ontario.

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PREAMBLE

This review was undertaken as part of Phase II of Professor Paul C. Weiler's study of the Ontario workers' compensation system. Its mandate was broad and its goals ambitious. This report attempts to provide the scientific background necessary to appreciate some of the complexities involved in compensating occupational diseases; it attempts to summarize the factors hindering the accurate assessment of the dimensions of the problem but, at the same time, given a broad perspective on the possible magnitude of the occupational disease problem; it tries to outline the major debates occurring in the medical-scientific community relevant to this area. Most importantly, the report attempts to describe the handling of occupational diseases by compensation systems generally and particularly by the Ontario Workers' Compensation Board.

Professor Weiler specifically requested that the study focus on the "serious" occupational diseases. Accordingly, acute diseases resulting from short-term exposures receive little attention. Time constraints and inadequacies in the data recording system of the WCB prevented a full discussion of non-malignant chronic diseases resulting from systemic exposure to toxic substances; and hazards to the reproductive system have been mentioned only in passing. Other conditions which affect large numbers of workers, such as "back disease," occupational dermatitis, hearing loss and stress-related diseases also receive disproportionately little commentary. The report does, however, provide a comprehensive review of the issues relating to occupational cancer and respiratory diseases. As it has been noted that cancer and lung disease are the only two major causes of death which have been steadily increasing, the decision to elaborate on these two groups of diseases and their occupational components was not arbitrary. Moreover, many of the problems identified in the adjudication of these claims do indeed apply to other categories of occupational diseases as well. In fact, the dilemma posed by these two categories of disease epitomizes the problems facing the compensation system.

The first chapter outlines the scope and possible dimensions of the occupational disease problem and provides an overview of how occupational diseases are handled by the compensation system. The following two chapters discuss occupational cancer and respiratory diseases respectively. Chapter IV focuses on the controversy regarding compensability of fatalities, as well as outlining the policy on adjudicating cardiac claims. Chapter V concludes the report with some reflections and recommendations.

Each of the major chapters (I, II, and III) consists of two parts. The first comprises a literature review outlining the state of knowledge at the time of the study (1981-1982) and the major points of debate in the field. It is intended to provide sufficient scientific background to allow the reader to appreciate the difficulties in claims adjudication and policy formulation. It is not intended to be a critical review of the literature and therefore does not attempt to pronounce judgement on the value of specific contributions to the body of scientific knowledge. The second part of each chapter reviews the development of policy at the Ontario Workers' Compensation Board and outlines the major issues of contention in this policy. A statistical overview is provided, procedural issues are discussed and cases are presented to illustrate the controversial overtones surrounding the adjudication of claims. Individual claim numbers, company names and the names of individual WCB staff physicians have been deleted to maintain confidentiality. Data from specific epidemiological studies relevant to policy formulation at the Board have been included in the appendices to facilitate the critical appraisal process which some readers may wish to undertake in greater depth.

The length of this report may prevent many readers from reading it through. Readers are therefore referred to the summary sections provided in the text which review the major issues discussed. It is intended that the detail provided and the depth with which the subject has been examined will make this document a useful reference on matters pertaining to the compensation of occupational diseases.

The staff of the Ontario Workers' Compensation Board have been of great assistance in conducting this study. The Board prepared background material and provided complete access to memoranda and internal studies. There was full co-operation in permitting the review of claim files upon request. The staff of the Statistical Services and Program Planning Branch of the WCB (now Program Planning Secretariat) deserves special mention for patiently conducting a sample study of some 400 industrial disease claims, extracting statistical and content information necessary for this report. They also provided a listing of all fatal claims for 1980. These surveys provided the major source from which the several hundred claims reviewed for this Inquiry were derived. Other sources from which the cases presented in this report were drawn included the listing in the Fatal Records maintained by the Industrial Disease and Dependents section of the Claims Adjudication branch, lists maintained by the Board doctors for their own records as well as those established at the request of this Inquiry, and cases brought to the attention of this Inquiry by injured workers and labour organizations. Case lists were, in fact, specifically requested from the latter groups to facilitate this study.

Lastly, the many others who contributed to this report deserve mention: Dr. James Nethercott, Director of University of Toronto's Occupational and Environmental Health Unit and the Occupational Health Clinic at St. Michael's Hospital, provided numerous insightful comments; Director of McMaster's Occupational Health Program, Dr. D.C.F. Muir, commented on the accuracy of some of the scientific material presented and offered his experience in this area; Dr. Harry Shannon reviewed the discussion of epidemiology in this report: Dr. Evert Nieboer and Dr. Sol Sax reviewed the sections on nickel and radiation respectively. Dorothy Wigmore and Claire Marie Fortin furnished important material, and Jerry Spiegel offered his assistance and support. The staff of the Canadian Centre for Occupational Health and Safety facilitated several literature reviews; Drs. John Chong, Ted Haines and other experts in the field of occupational health contributed their valuable comments. Important insights were derived from the many briefs submitted to the Weiler Inquiry by various individuals and organizations, and the disabled workers or their survivors who made the effort to present their concerns certainly had a profound impact.

The staff of the Ministry of Labour must be acknowledged for their patience in the processing of this report. Catherine Wilbraham and Frank Krauss designed the format and prepared the layout of the tables and text. Cherith Muir undertook the final editing and organized the publication of this manuscript. Mary O'Donnell co-ordinated efforts, and Accentus Word Processing did the typing. Any opinions and/or errors in the text, are however, the responsibility of the author. Research in the preparation of this study was undertaken in the period 1981-82.

Criticism of current policy and procedure at the Ontario Workers' Compensation Board is not intended to imply ill-will on the part of specific individuals. Rather it attests to the serious need for careful public consideration of the philosophical underpinnings of the current system. Ontario has played an historical role in compensation legislation. It is therefore not inappropriate that Ontario take steps to usher in yet another new era. It is hoped that this report will contribute to the development of a system that better provides proper redress to the unfortunate victims of occupational disease.

TABLE OF CONTENTS

CHAPTER 1 - OCCUPATIONAL DISEASE AND COMPENSATION: AN OVERVIEW

PART A. SCOPE AND DIMENSIONS OF OCCUPATIONAL DISEASE 1

1. **Introduction** 1

2. **The Scope of Occupational Diseases** 2

   2.1 Physical Agents 3
   2.2 Biological Hazards 3
   2.3 Chemical Agents 3
   2.4 Ergonomic Factors 6
   2.5 Psychosocial Stress 7

3. **Dimensions of Occupational Disease** 8

   3.1 Difficulties in Measuring the Occupational Disease Problem 8
   3.2 Estimates of the Dimensions of Occupational Disease 14

4. **The Issues Summarized** 19

References and Notes 20

Appendices:

APPENDIX 1 Criteria of Health Impairment 22

APPENDIX 2 "Occupational Disease is Grossly Under-Reported:" A Look at the Discher Study 24

APPENDIX 3 Data about Exposure in Ontario 28

APPENDIX 4 Results from Survey of Disabled and Non-Disabled Adults 29

PART B. COMPENSATING OCCUPATIONAL DISEASES 30

1. **Facts and Figures** 30

2. **The Decision-Making Framework in Various Jurisdictions** 37

   2.1 Approaches to Determining the Work-Relatedness of Disease 37
   2.2 Ontario 37
   2.3 Canada 38
   2.4 U.S. 39
   2.5 Europe 40
   2.6 Comparative Assessment 42

3. **The Problems Introduced** 44

   3.1 Degenerative Disc Disease and Heavy Labour 44
   3.2 Chronic Poisoning from Chemical Exposure 46
   3.3 Noise-Induced Hearing Loss 48
   3.4 Diseases Induced by Occupational Stress 50

4. **The Issues Summarized** 52

References 53

APPENDIX 1 Morbidity and Mortality - Ontario 56
APPENDIX 2 Compensated Injuries - Other Jurisdictions 58
APPENDIX 3 Schedule III - Ontario Workers' Compensation Act 60
APPENDIX 4 Occupational Factors in Degenerative Disc Disease 61
APPENDIX 5 Claims for Noise-Induced Hearing Loss 64
APPENDIX 6 Occupational Stress and Cardiovascular Disease 66

CHAPTER II - CANCER AND THE WORKPLACE

PART A. ISSUES AND DIMENSIONS OF OCCUPATIONAL CANCER 73

1. Cancer in Ontario - Facts and Figures 73

2. Current Knowledge About Cancer Causation 81

2.1 The Nature of Cancer and Carcinogenesis 81
2.2 Agents Capable of Causing Cancer 83
2.3 Factors Influencing the Latency Period 85
2.4 The Multifactorial Nature of Carcinogenesis 88
2.5 The Threshold Debate 89
2.6 Multiple Causes - Single Disease 93

3. Difficulties in Assessing Carcinogenicity 93

3.1 Limitations of Epidemiology 93
3.2 Animal Studies and Short-Term Tests 96
3.3 Internationally Recognized Human Carcinogens 98

4. Exposure Estimates and Other Data Sources

4.1 Estimates of the Extent of Exposure - The NIOSH Attempt 100
4.2 Occupational Health Data Sources in Ontario 104

5. Estimates of the Fraction of Cancer Related to Workplace Factors

5.1 Inherent Uncertainties 106
5.2 The Different Approaches and Range of Opinions 110
5.3 The Debate Surrounding the Estimates Paper 113
5.4 Implications of the Production Pattern of Possible Carcinogens 114

6. The Issues Summarized 115

References 119

Appendices

APPENDIX 1 Established Human Carcinogenic Agents 124
APPENDIX 2 Latent Periods in Human Occupational and Environmental Cancer 125
APPENDIX 3 Evaluating Carcinogenic Risk and Known Occupational Carcinogens. (IARC Methods and Findings) 130
APPENDIX 4 Proportion of Cancer Attributed to Various Different Factors by Various Authors 137

PART B. OCCUPATIONAL CANCER AND THE ONTARIO WORKERS' COMPENSATION SYSTEM

1. Historical Role and Development of Guidelines 142
    - *1.1 Historical Role Played by the Ontario Workers' Compensation Board* 142
    - *1.2 The Development of Policy* 144

2. Facts, Figures and Issues 146
    - *2.1 The Volume of Cancer Claims* 146
    - *2.2 Rejection Rate of Occupational Cancer* 149
    - *2.3 Rejection Rate and Guidelines* 149
    - *2.4 The Appeal Process* 156
    - *2.5 Under-Reporting of Occupational Cancer* 157
    - *2.6 The Delay Factor* 165
    - *2.7 Scientific Evidence and Medical Knowledge* 165
    - *2.8 Outreach Programs* 169

3. The Decision-Making Process in the Adjudication of Cancer Claims 170
    - *3.1 Radiation and Cancer* 170
    - *3.2 Cancers Caused by Soots, Tars and Oils* 198
    - *3.3 Cancer and Nickel Refining* 226
    - *3.4 Asbestos-Related Cancers* 242
    - *3.5 Cancer Caused by Other Substances* 255

4. The Issues Summarized 257

References 260

Appendices:

APPENDIX 1 B.C. Schedule of Diseases Re: Cancer 271

APPENDIX 2 Uranium-Lung Cancer Claims 272

APPENDIX 3 Data About Polycyclic Aromatic Hydrocarbons (including Coke Oven and Foundry Studies) 274

APPENDIX 4 Data From Mortality Studies of Nickel Refinery Workers in Ontario 286

APPENDIX 5 Guidelines for the Allowance of Asbestos-Related Cancers and Studies of Occupational Laryngeal Carcinoma 288

CHAPTER III - NON-MALIGNANT OCCUPATIONAL RESPIRATORY DISEASE

PART A. ISSUES AND DIMENSIONS OF CHRONIC OCCUPATIONAL RESPIRATORY DISEASE 294

1. Introduction 294
    - *1.1 Overview* 294
    - *1.2 Historical Background* 295

2. An Overview of Respiratory Disease 295

2.1 Prevalence of Respiratory Disease 295
2.2 Structure and Function of the Respiratory System 296
2.3 The Deposition and Clearance of Dust from the Lungs 301
2.4 Pathological Reactions of the Lung 305

3. The Nature and Prevalence of Occupational Lung Diseases 310

3.1 Pneumoconiosis 310
3.2 Chronic Obstructive Lung Diseases 329

4. Issues of Diagnostic Accuracy and Disability Assessments 342

4.1 Evaluation of the Impairment and Disability Attributable to Respiratory Disease 342
4.2 Chest X-Rays: Intra- and Inter- Reader Variability 348
4.3 Limitations in the Interpretation of Pulmonary Function Tests 350
4.4 "Subjective" Symptoms and "Objective" Testing 351

5. The Issues Summarized 353

References and Notes 358

Appendices:

APPENDIX 1 Respiratory Disease Mortality Trends in Canada (1969-78) 367

APPENDIX 2 The Incidence and Severity of Pneumoconiosis in Canada 369

APPENDIX 3 Labour Force by Selected Industries in 1971 and Occupations at Risk of Asbestos Exposure 371

APPENDIX 4 Pneumoconiosis Across Canada 373

APPENDIX 5 Incidence of Radiological Silicosis 375

APPENDIX 6 Chronic Obstructive Lung Disease (Ottawa and Sudbury) 379

APPENDIX 7 Chronic Obstructive Lung Disease - The Experience in One Converter Plant 380

APPENDIX 8 Chronic Bronchitis and Pulmonary Impairment - Elliot Lake Mining Operations 383

APPENDIX 9 Comparison of Pulmonary Impairment Rating Schemes 386

APPENDIX 10 Variability in Interpretation of Pulmonary Function Tests 389

PART B. THE COMPENSATION OF OCCUPATIONAL RESPIRATORY DISEASES

1. The Facts, Figures and Issues Involved 390
   - 1.1 *Identifying Work-Related Diseases and the Work-Relatedness of Disease* 390
   - 1.2 *Disability Assessments* 406

2. The Adjudication of Pneumoconiosis Claims 414
   - 2.1 *Evolution of Policy* 414
   - 2.2 *Claim Procedure in the Adjudication of Pneumoconioses* 416
   - 2.3 *Case Presentations - Disability Assessment in the Presence of Confounding Factors and Eligibility for Rehabilitation* 426

3. The Adjudication of Chronic Obstructive Lung Disease Claims 432
   - 3.1 *Policy on the Adjudication of Asthma Claims* 432
   - 3.2 *Policy on the Adjudication of Pulmonary Emphysema* 447
   - 3.3 *Policy on the Adjudication of Chronic Obstructive Lung Disease Claims* 456

4. The Issues Summarized 466

Appendices:

APPENDIX 1 Summary of the Evolution of Board Policy - Silicosis and Other Pneumoconioses 469

APPENDIX 2 Pulmonary Function Tests and Disability Assessment 472

APPENDIX 3 WCB Policy and Revised Policy Concerning Allergic Respiratory Disease 473

APPENDIX 4 Guidelines for Adjudicating Pulmonary Emphysema due to Alpha-1-Antitrypsin Deficiency 476

APPENDIX 5 Guidelines for Adjudicating Chronic Obstructive Lung Disease Claims in Smelter Workers 479

CHAPTER IV - WORK-RELATED DEATH: THE ADJUDICATION OF CLAIMS FOR SURVIVOR'S BENEFITS AT THE ONTARIO WORKERS' COMPENSATION BOARD

1. An Analysis of Death Benefit Statistics 481
   - 1.1 *Overview* 481
   - 1.2 *Compensation Award at Time of Death as Predictor of Outcome of Claims for Survivor's Benefits* 483
   - 1.3 *Outcome of Claims for Survivor's Benefits by Compensable Condition* 486
   - 1.4 *Outcome of Claims for Survivor's benefits by Cause of Death* 486
   - 1.5 *Cause of Death by Compensable Condition* 489
   - 1.6 *Commentary* 492

2. Controversy Over Cause of Death in Workers Who Were Disabled by Occupational Respiratory Disease 493

  2.1 *Death from Respiratory Disease* 493
  2.2 *Cardiac Death Among Workers with Occupational Respiratory Disease* 498
  2.3 *Other Causes of Death Among Workers with Industrial Lung Disease* 502

3. The Compensation of Cardiac Disease 504

  3.1 *Statistics* 504
  3.2 *Policy* 505
  3.3 *Evolution of Guidelines* 506
  3.4 *Case Presentations* 507

4. The Issues Summarized 513

Appendices:

APPENDIX 1 Cardiac Claims - Guidelines for Adjudication 516

CHAPTER V - REFLECTIONS AND RECOMMENDATIONS

1. Reflections on the Compensation of Industrial Diseases 519

2. Recommendations on Matters External to the WCB 522

  2.1 *On the State of Medical Knowledge and the Organization of Occupational Medicine Services* 522
  2.2 *The Maintenance of Exposure Records* 524
  2.3 *Responsibility for Research* 525
  2.4 *Data Collection and Occupational Disease Registries* 525
  2.5 *Outreach* 526

3. Recommendations Regarding WCB Policy and Procedure 527

  3.1 *Policy Formulation* 527
  3.2 *Issues Pertaining to Claims Procedure* 531

4. Summary 532

LIST OF TABLES AND FIGURES

Table/Figure Number	Title	Page
IA-1	Factors Capable of Causing Occupational Disease	5
IB-1	Total Allowed Claims and Allowed Industrial Disease Claims, WCB (Ontario) (1976-1980)	31
IB-2	Occupational Disease Awards Ontario WCB, 1980	33
IB-3	Occupational Disease Awards - B.C. and Ontario	34
IB-4	Cancer and Workers' Compensation Across Canada	36
IB-5	Workers' Compensation for Disabling Occupational Diseases in Various Jurisdictions	43
Fig.IIA-1	Age Adjusted Cancer Mortality Rates by Sex - Ontario (1940-76)	74
Fig.IIA-2	Age Adjusted Cancer Mortality Rates per 100,000 - U.S. (1900-1980)	74
IIA-1	Total Deaths with Mention of Cancer - Ontario, 1979	75
IIA-2	Cancer Mortality Rates and Cancer Deaths As Proportion of Total Deaths by Sex - Ontario (1940-76)	76
Fig.IIA-3a	Age Adjusted Mortality Rates - Selected Male Cancer Sites - Ontario (1931-75)	78
Fig.IIA-3b	Age Adjusted Mortality Rates - Selected Female Cancer Sites - Ontario (1931-75)	78
IIA-3	Age Adjusted Incidence Rates for Selected Cancers by Sex - Ontario (1966 and 1971)	79
IIA-4	Average Percentage of Change Age-Adjusted Cancer Mortality and Incidence Rates (1969-76)	79
IIA-5	Changes in U.S. Cancer Incidence Rates (1970-75)	80
IIA-6	5-year Survival Rates - Major Cancer Sites at All Stages (1965-69)	80
IIA-7	Estimated Proportion of Cancer Incidence by Site and Sex	83
IIA-8	A Comparison of Biological Properties of Initiating and Promoting Agents	87
Fig.IIA-4	A Simplified Scheme for the Steps in the Induction of Cancer by a Chemical	87
Fig.IIA-5	Diagrammatic Representation - Dose-Response Relationships	91

Table/Figure Number	Title	Page
Fig.IIA-6	Possible Effect of a Small Dose of Carcinogen on Mortality With and Without Background Levels	91
IIA-9	IARC Groupings of Carcinogens and Probable Carcinogens (1980)	101
Fig.IIA-7	Annual Production - All Synthetic Organic Chemicals	116
Fig.IIA-8	Annual Production - Selected Carcinogens	116
IIB-1	Historical Development of WCBO Cancer Policy	143
IIB-2a	All Allowed Compensation Claims, Industrial Disease Claims and Cancer Claims 1974-80	147
IIB-2b	All Allowed Fatal Claims, Fatal Disease and Fatal Cancer Claims	147
IIB-3	All Fatal Claims Reported (1980) by Status and Cause	148
IIB-4a	Cancer Claims Reported to WCBO (1980 and 1981) by Status and Cancer Type	151
IIB-4b	Fatal Cancer Claims Reported (1980) by Status and Cancer Type	151
IIB-5	Cancer Claims Included in the Industrial Disease Survey by Last Claim Status	152
IIB-6	Total Rejected Claims	153
IIB-7	Allowed (and Rejected) Cancers by Site and Exposure	154
IIB-8	Cancers Reported by Site and Exposure Substance	155
IIB-9	Cancer Claims in the Industrial Disease Survey by Exposure and Last Claim Status	156
IIB-10	Cancer Claims in the Industrial Disease Survey by Outside Group Involvement and First Claim Status	158
IIB-11a	Cancer Claims in the Industrial Disease Survey by Person/Group Who Filed Claim and Claim Status	159
IIB-11b	Outcome of Claims Initiated by Employer or Health Professionals Compared to Those Initiated by Claimant or Union	159
IIB-12a	Claims Review Branch. Adverse Decisions and Objections, Total Claims Reviewed (1979, 1980)	160
IIB-12b	Claims Review Branch Adverse Decisions and Objections, Cancer Claims in Sample Survey	160

Table/Figure Number	Title	Page
IIB-13a	Appealed Claims by Last Outcome - Total Claims (1980)	161
IIB-13b	Appealed Cancer Claims by Last Outcome - Sample Survey	161
IIB-14	Cancer in Ontario That Definitely Can Be Produced by Occupational Hazards	162
IIB-15	Cancer Reported to WCB as a Percentage of Estimated Total Occupational Cancers in Ontario by Cancer Site	164
IIB-16	Types of Cancer in Asbestos-Related Claims (Indexed)	166
IIB-17	Claims Processing Times - All Claims	166
IIB-18a	Frequency Distribution of Cancer Claims in the Industrial Disease Survey by Weeks From Date Claim Set Up to Date Initial Decision Received and First Claim Status	167
IIB-18b	Frequency Distribution of Cancer Claims in the Industrial Disease Survey by Weeks from Initial Decision to Date Conveyed to Claimant and First Claim Status	168
IIB-19	Cancers Definitely and Probably Caused by Radiation	174
IIB-20	Ontario Workers' Compensation Board Lung Cancer Claims - Radon Gas Exposure	182
IIB-21a	Lung and Sinus Cancers Allowed and Rejected Copper Cliff and Port Colborne	240
IIB-21b	Exposure Time of Claims Denied Due to Insufficient Exposure	240
Fig.IIIA-1	The Respiratory System	298
Fig.IIIA-2a	Lung Volumes and Spirometric Tracings	300
Fig.IIIA-2b	Forced Expiratory Volumes	300
Fig.IIIA-3	Dust Deposition and Particle Size	303
Fig.IIIA-4	Spectrum of Respiratory Pathology	306
Fig.IIIA-5	Cumulative and Surviving Number of Silicotics (1925-81)	320
Fig.IIIA-6	Age, Time of First Exposure and Disability at Time of First Rating for Silicosis (1925-81)	322
IIIA-1	Mortality Among Miners Receiving Workers' Compensation for Silicosis	323

Table/Figure Number	Title	Page
IIIA-2	Mortality Among Asbestotics	324
Fig.IIIA-7	Age at Recognition, Age at Death, Number of Totally Disabled	325
Fig.IIIA-8	Test-Schema for Determining Severe Respiratory Impairment	347
IIIB-1	Sequence of Events in Claim's Adjudication - Respiratory Diseases	391
IIIB-2	Silicosis and Asbestosis Claims by Person/Group Who Filed First Claim	393
IIIB-3	Allowed Compensation Claims for Non-Malignant Respiratory Disease, 1980	394
IIIB-4a	Claims Received for Respiratory Conditions by First Claims Status and Person/Group Who Filed Claim	395
IIIB-4b	Outcome of Silicosis Claims Initiated by Employers or Health Professionals Compared to Claims Initiated by Claimant or Union	395
IIIB-5	Allowed vs. Rejected Non-Malignant Respiratory Disease Claims at Ontario WCB Reported by Several Sources	397
IIIB-6	Claims Received for Silicosis by Outside Group Involvement and First Claim Status	399
IIIB-7	Frequency Distribution of Claims Received for Silicosis by Weeks From Date Claim Initiated to Date Initial Decision Reached and First Claim Status	400
IIIB-8	Frequency Distribution of Initially Rejected Claims by Weeks from Referral to Appeal to Decision Date, Type of Industrial Disease and Number of Appeals	402
IIIB-9	Frequency Distribution of Silicosis Claims by Weeks From Completion of Primary Adjudication to Claimant Notification and First Claim Status	403
IIIB-10	Initially Rejected Claims for Silicosis by Appeal Process and Last Outcome	404
IIIB-11	Claims Received for Silicosis by Outside Group Involvement and Last Appeal Outcome	405
IIIB-12	Frequency Distribution of P.D. Claims in the Silicosis Index by Diagnosis and % P.D., Award to Date	407
IIIB-13	Compensation Paid or Allowed by Type of Benefit Paid - Silicosis Claims Set up Prior to July 1, 1979	411

Table/Figure Number	Title	Page
Fig.IIIB-1	Claims Adjudication - Industrial Diseases	415
IIIB-14	Guidelines in Differentiation of Stable Obstructive Airways Diseases	453
IV-1	1980 Survivor's Benefit Claims by Claim Status and Category	482
IV-2	1980 Survivor's Benefit Claims by Permanent Disability Award and Status	484
IV-3	Survivor's Benefits Claims January 1, 1981 - June 30, 1981 by Decision About Work-Relatedness	485
IV-4	1980 Survivor's Benefit Claims by Permanent Disability Award, Compensable Condition and Claim Status	487
IV-5	1980 Survivor's Benefit Claims by Permanent Disability Award, Cause of Death and Claim Status	488
IV-6	1980 Death Benefit Claims by Cause of Death, Compensable Condition and Claim Status	490

CHAPTER I

OCCUPATIONAL DISEASE AND COMPENSATION: AN OVERVIEW

A. THE SCOPE AND DIMENSIONS OF OCCUPATIONAL DISEASE

1. Introduction

For centuries it has been known that various conditions of work can adversely affect health. By 1713 Bernardo Ramazzini, the "father of occupational medicine", had described many occupational diseases related to physical, chemical and biological agents in the workplace. Even the importance of ergonomic factors was alluded to at that time. Ramazzini referred not only to "the injurious character of the materials handled" but also to "certain violent and irregular motions and unnatural postures of the body by which the internal structure is impaired." He was well aware of industrial deafness, of the hazards of radiation, and he clearly appreciated the insidious nature of many substances in the work environment.[1] By 1775, Percival Pott had linked the scrotal cancer in chimney sweeps to their occupation and by the turn of the century many occupational carcinogens had already been documented. Occupational lung diseases have been well known for centuries as has been systemic poisoning from heavy metals. Liver damage and death from exposure to solvents such as chloroform was recorded by 1894, and by 1920 death from chronic benzene exposure was acknowledged. The hazards of asbestos were so well documented that by 1918 insurance companies were refusing to sell life insurance to asbestos workers.

While occupational disease is not a newly uncovered problem, its potential magnitude has only recently been widely recognized. The increasing awareness of occupational health and safety, particularly in the labour movement, has led to pressure for regulating industrial health hazards and to efforts for cleaning up the workplace. It has also led to questions about the adequacy of redress currently provided by workers' compensation for occupational disease victims.

While there is still some debate regarding the purpose of workers' compensation, it is now generally accepted that the goals of the workers' compensation system include:

(1) the provision of adequate income maintenance for workers disabled by occurrences arising out of and in the course of employment;
(2) the assurance that such workers receive adequate health care and rehabilitation services; and
(3) the structuring of the system so that it encourages the maintenance of a safe and healthful workplace.

Several U.S. studies have already underlined the inadequacy of their compensation system in meeting these goals for victims of industrial diseases. Based on the Interim Report to Congress on Occupational Diseases[2], it has been concluded, first, that workers disabled from occupational disease receive less than an equitable trade-off when they give up their legal rights to sue employers for work-related illnesses in exchange for workers' compensation benefits; secondly, there does not appear to be an effective mechanism for swiftly delivering benefits to occupational disease victims; thirdly, considering the few occupational disease victims who receive benefits and the size of the payments involved, the system does not provide much of an incentive to reduce future exposure to industrial health hazards; and fourthly, the costs of compensation and medical care are being passed on to other programs such as social security and welfare as well as to the families of disabled workers themselves.[3]

The compensation system in Ontario differs in many respects from those existing in the U.S. Extensive litigation and frequent use of 'out of court' settlements for example, are not present in the Ontario system. Nonetheless, many of the factors leading to the failure of the U.S. compensation system to deal effectively with occupational diseases are also operative in Ontario and there is reason to suspect that the above conclusions may well be applicable here as well. Some of the reasons pertain to the very nature of occupational disease and the climate of controversy which surrounds it.

There is disagreement over the definition of "occupational disease" with respect to what constitutes "occupational" as well as what constitutes "disease." Controversy as to the work-relatedness of disease stems partly from the multifactorial nature of many, if not the vast majority, of diseases. Etiological difficulties are compounded by diagnostic difficulties, further compounded by the inadequacy of medical training in the area. The paucity of research pertaining to industrial hazards and the generally poor state of data collection regarding exposure, contribute significantly to the difficulties in recognizing occupational disease and adjudicating occupational disease claims.

Difficulties in distinguishing work-related from non-work-related diseases are matched by difficulties in determining the borderline between health and disease. With the availability of ever more sophisticated measures of health, debate focuses on what extent of "deviation from normal" constitutes disease, as well as the very definition of "normal" itself. Some argue that compensation should be provided only for "disability" measured by impaired earning potential; others argue that any functional impairment is unacceptable and should be compensated.

The difficulties in defining occupational disease have their consequences in the compensation system. They also greatly affect the ability to even evaluate the extent of occupational disease and hence to assign appropriate resources to its prevention. The area of occupational health has thus become a microcosm of conflict between labour and management, and workers' compensation has become an increasing source of controversy.

Part A of this chapter will discuss the scope and dimensions of various occupational diseases and the issues behind these assessments. Part B will provide an overview of the various approaches to compensating victims of occupational disease and the experience of the Ontario WCB in handling these claims. Difficulties associated with the adjudication of various categories of diseases will be briefly illustrated.

2. The Scope of Occupational Diseases

With the increasing complexity of modern industrial society and as knowledge of health effects continues to accumulate, occupational diseases take on an ever more varied nature. They may be acute, arising soon after exposure, such as may occur from poisonous fumes or gases; they may be chronic, developing slowly over time, such as silicosis or noise-induced hearing loss, or they may occur only after a long latency period, as is the case for occupational cancer. The effect of the exposure may be local, affecting the part of the body directly exposed, such as the skin, eyes, or mucous membranes of the respiratory tract;

or it may be systemic in nature, occurring when the chemical is absorbed into the blood, affecting various internal organs of the body. Disease may occur in the exposed individual or it may appear in subsequent generations as a result of alterations in the genetic apparatus, for example, from the exposure to radiation. Moreover, one agent may cause a variety of different occupational diseases depending on the dose intensity and rate of exposure. Not only do physical, chemical and biological agents affect workers' health, but ergonomic factors and psychosocial stresses have taken on increasing importance. Table IA-1 outlines some of the health hazards included in each of these categories.

2.1 *Physical Agents*

Physical agents capable of producing adverse health effects include temperature factors such as heat, cold and sudden changes in temperature, as well as changes in pressure, (capable of producing decompression sickness or "the bends"). Noise-induced hearing loss and vibration-induced white finger are well recognized occupational diseases; and studies on the role of noise and vibration in a wide variety of diseases are accumulating. The hazards of electricity are well-known, as are many of the potential effects of non-ionizing radiation, such as ultraviolet light, infra-red radiation and microwaves, as well as ionizing radiation, such as x-rays, gamma rays, radon, radon gas and radon daughters.

2.2 *Biological Hazards*

Biological agents including plants, animals, bacteria, viruses, parasites, toxins, venoms and various organic materials may result in infection, infestation, bites or allergies. Measles contracted by a teacher from a student, or hepatitis contracted by a hospital worker from inadequate sanitary procedures or ailments more obviously linked to occupation such as "farmer's lung", or "grain-handler's disease" are all examples of occupational diseases resulting from exposure to biological agents. Many of these diseases tend to be more easily identifiable in origin compared to other occupational diseases and generally constitute a fair percentage of the disease claims in workers' compensation statistics as illustrated in Part B of this chapter.

2.3 *Chemical Agents*

Many thousands of chemical agents are currently in use and are collectively responsible for the majority of presently recognized occupational diseases. These agents may take the form of dusts, fumes, gases and vapours. Their chief method of entering the body is by inhalation, but some chemicals may be absorbed through the skin. Ingestion may occasionally be a factor especially where smoking and eating occur in areas where toxic agents are handled.

The extent of biological damage resulting from dust exposure depends on the size of the particle, the concentration in the breathing zone, the length of exposure, the rate and depth of breathing during the exposure, the composition of the dust itself and its rate of elimination from the body. Dusts and fumes may be inert; they may cause disease in the respiratory system or they may be systemic poisons. Even the so-called "harmless" or nuisance dusts such as iron, aluminum and titanium, do have some harmful effects and certainly heavy exposure may cause irritation to the nose and throat.

Some of the most important dusts among those known to cause lung disease are silica (causing silicosis), asbestos (causing asbestosis and cancer), coal dust (causing coal workers' pneumoconiosis), beryllium (causing berylliosis) and cotton dust (causing byssinosis). Silica, found most commonly in mining and foundry work, has been the largest source of fibrosing (or scarring) occupational lung disease in Ontario. Asbestos has also been the focus of considerable concern in this province.

Dusts and fumes may also be poisonous to organs other than the lungs, as are many gases and vapours. Exposure to lead, for example, usually occurs by inhalation, but some lead may be ingested and tetraethyl lead readily penetrates intact skin. Lead accumulates in the body and even a small amount inhaled over a prolonged period may result in insidious lead poisoning even well after exposure has ceased. Lead poisoning may be characterized by impairment of the neurological system resulting in symptoms such as headaches, weakness, depression, insomnia, inability to concentrate and at more serious exposures, lead palsy and encephalopathy (diffuse disease of the brain tissue); it may affect the hematopoeitic (blood-cell producing) system, causing anemia; it may affect the kidneys, it may cause abdominal pain, it may affect the reproductive system, and so on. Another in the category of toxic fumes and dusts is arsenic, which in addition to its generally poisonous properties and carcinogenicity, can produce skin irritation, pigmentation and wart-like growths. Cadmium fumes, also possibly carcinogenic, are very injurious to the lungs and can produce pulmonary edema within hours of exposure. Many metal fumes, particularly zinc, can produce a febrile flu-like condition called "metal fume fever."

Some poisonous gases and vapours are odourless, colourless and non-irritating, while others may easily be identified by the senses. The former group includes carbon monoxide, hydrogen cyanide, mercury vapour and others. Carbon monoxide, ubiquitous in industry, is found wherever there is incomplete combustion. Its toxic effects are well known. Hydrogen cyanide has been a cause of death when used for fumigation. Acute exposure to high concentrations of mercury may harm the gastrointestinal system causing abdominal pain, vomiting, and diarrhea; it may cause inflammation of the lungs, kidney damage and even circulatory and respiratory failure. Organic mercurials may produce tremors, personality changes (as occured in the "Mad Hatter"), visual field restrictions and kidney damage. Chronic exposure to organic mercurials may also produce ataxia (unstable gait), paresthesia (abnormal sensations), tremor, emotional instability, slurred speech and even impaired hearing. Some gases which give no warning of their presence may act as asphyxiants and cause harm by relatively reducing the available oxygen. These include nitrogen, argon, helium and carbon dioxide.

Other gases and vapours can be seen. Nitrous fumes, for example, consisting of mixtures of oxides which are brownish in colour, may be slightly irritating but may unknowingly be breathed in concentrations sufficient to be fatal. Some gases and vapours are extremely irritating. Sulfur dioxide, chlorine, ammonia and hydrochloric acid vapours are responsible for a high incidence of acute respiratory disease and prolonged exposure may lead to chronic obstructive lung disease. Ozone is primarily an irritant to the eyes and mucous membranes, but may produce pulmonary edema if exposure is severe enough. It may also cause headache, malaise and fatigue.

Table IA-1

Factors Capable of Causing Occupational Disease

Physical Agents	Biological Agents	Chemical Agents	Ergonomic Factors	Psychosocial Factors
Temperature heat, cold, sudden changes Barometric Pressure Vibration air vibration (noise), mechanical vibration Electricity Radiation ionizing radiation (x-rays, gamma rays radioactive substances), non-ionizing radiation (UV light, IR, microwaves)	Animals Plants Microorganisms bacteria viruses, etc. Fungi Venoms, toxins Materials (dusts, etc.)	Dusts Fumes Vapours Gases Liquids	Workplace Design layout, traffic flow, mobility, etc. Lighting, sound temperature Process and equipment design Person/job fit	Nature of Occupation knowledge and technical requirements societal responsibility, self-perceived status, social isolation, etc. Job Characteristics decision-making participation resource availability, support from co-workers and supervision, workload, responsibility for others threat of disciplinary actions Job Conditions job security, work hours, wages and benefits, etc.

Many solvents, dilutents, cleaning agents, adhesives and paints used in industry contain volatile substances recognizable by their odour. Some are irritating or have an anesthetic property when encountered in high concentrations, but small amounts encountered over a prolonged period may also produce a variety of different serious diseases. Toluene, for example, a substance which may be encountered in glues, paints, varnishes, enamels and in the manufacture of chemicals, is an irritant to the skin and mucous membranes. High concentrations may initially produce central nervous system symptoms such as headache, dizziness and fatigue which may be followed by convulsions and death. Chronic low level exposure, however, may insidiously produce liver damage. Moreover, often small quantities of benzene may be found in toluene preparations which may result in changes in the bone marrow and blood, decreasing the worker's resistance to infection or ultimately producing serious diseases such as leukemia or aplastic anemia. Inhalation of moderate concentrations of carbon tetrachloride, for example, may cause nausea and vomiting and result in liver and kidney damage. Another of the chlorinated hydrocarbons, trichloroethylene, is a very commonly used degreasing agent. It has anesthetic properties, and although less toxic than carbon tetrachloride, may have many of the same properties and is a suspected carcinogen as well. Other solvents may promote neurological disorders, arteriosclerosis and heart disease. One such substance, carbon disulphide, is used in rubber-lining adhesives in the manufacture of rayon.

Occupational dermatitis (skin disease) is one of the most easily recognized and commonly reported effects of chemicals. However, not only can these chemicals irritate the skin but they may be absorbed into the blood stream to produce a wide variety of acute effects as well as serious chronic diseases. Toxic materials, in addition to being poisonous, may also be carcinogenic (capable of causing cancer), mutagenic (capable of causing a genetic change transmissable to the offspring) or teratogenic (capable of causing a birth defect).

There are some 12,000 listed toxic materials in the U.S. As illustrated above, these can cause diseases of many different types and of many different organ systems of the body. Knowledge of the possible effects of the various chemicals singly or in combination is only beginning to accumulate. Many of the diseases caused by these exposures are indistinguishable from "ordinary diseases of life", i.e. those not related to the workplace, thereby clouding the recognition of occupational causation. It is no wonder that the compensation of workers who suffer ill effects from these exposures is a difficult task, as will be explored in Part B of this chapter.

2.4 *Ergonomic Factors*

Ergonomics refers to the body of scientific and technical knowledge on the relationship between the worker and his or her work. It comprises knowledge regarding energy expenditure and environmental stresses, biomechanics, sensorimotor tasks and controls. It is often used synonomously with the term "human factor engineering", defined as the application of information about human characteristics, capacities and limitations to the design of machine systems and environments so that people can work safely, comfortably and effectively. Chronic degenerative diseases of the musculoskeletal system have been attributed to faulty design of the work-site and equipment and the characteristics of the task. Tenosynovitis, or inflammation of the tendon sheath around the wrist joint, for example, may occur because of repetitive wrist movements; backache from vertebrae degeneration as a result of heavy lifting is also well-established. Headache and eyestrain have been related to lighting factors, and are a particular concern with the increasing use of video display terminals (VDTs). Other factors include the basic layout, traffic flow, temperature and activity level demands of a job. A worker may suffer a heart attack if, for example, the environmental stresses and particularly the exertional demands of the job are beyond the capabilities of his cardiovascular reserve.

2.5 *Psychosocial Stress*

The term "occupational stress" refers to stimuli that can disrupt the worker's physiological or psychological well-being. The origin of the stress may be a physical factor, such as heat or noise stress, or it may be caused by psychological or psychosocial factors. The alienation resulting from repetitive and fragmented mass production jobs, the rate and tempo of production, the working hours, the length of the work day, the methods of remuneration, the exercise of authority, role conflict and ambiguity, social isolation and many other factors involved in the organization of work may well be important determinants of a worker's physical well-being as well as mental health.

Compensation has begun to be awarded in various jurisdictions for disease resulting from psychological stress. A compensation claim was recently won by a worker in Quebec for a nervous condition resulting from harassment by a member of management, or what Steelabour called "foremanitis." (Part B of this chapter discusses this issue.)

Thus it can be seen that workplace factors may affect every aspect of a worker's health. The diseases that ensue may result from a wide variety of agents or working conditions, and each of these agents or conditions may result in a host of different diseases depending on numerous characteristics of the exposure and the individual affected. A small dose may produce one effect whereas a larger dose may produce quite a different one. For example, a small dose of carbon monoxide may induce drowsiness or chest pain in certain individuals, but a larger dose can cause death. Moreover, the level of exposure needed to produce chronic effects is usually very much less than the minimum amount required to demonstrate an immediate or acute effect; and the mechanisms for the acute and chronic damage may be very different and may manifest harm in totally different organ systems of the body. For example, many solvents may have an acute effect on the nervous system, causing drowsiness, whereas chronic exposure to low levels of various solvents may cause liver or kidney damage or even cancer.

Furthermore, occupational diseases often result not from a single agent but from a combination of factors. It is well known that different chemicals may interact synergistically (i.e. in greater than an additive fashion) to produce disease. In fact, hazards of physical, chemical, biological, biomechanical and psychological origin may interact together to produce disease. Carbon monoxide and heat, for example, may have a synergistic effect on the cardiovascular system. In combination with exertional demands or the stress of disciplinary pressure on the job, devastating cardiovascular disease may result.

The broad scope of occupational disease poses great difficulties for the compensation system, which, to be faithful to its mandate must take into consideration a wide variety of workplace factors in the adjudication of claims. The delineation of occupational from non-occupational disease is hence subject to much controversy. The issues creating and perpetuating these difficulties affect not only the ability to adjudicate claims, but also the ability to accurately appraise the dimensions of the occupational disease problem in our society. This aspect will be elaborated upon in the next section.

3. Dimensions of Occupational Disease

Difficulties in distinguishing occupational from non-occupational disease, lack of consensus regarding the definition of "disease" itself, the poor state of medical knowledge and exposure records, the organizational structure of occupational medicine services, and the dearth of scientific research on occupational disease, all contribute to the inability to quantify the occupational disease problem. Moreover, the nature of occupational diseases is not static, but changes along with changes in the nature of production. These factors will be reviewed below.

3.1 Difficulties in Measuring the Occupational Disease Problem

3.1.1 Etiological Difficulties - Distinguishing Occupational from Non-Occupational

Not only do occupational diseases cover an immensely broad scope and have numerous varied manifestations, but there are few diseases that are associated only with a work-related activity or exposure. Although there are some diseases that are specifically occupational, silicosis or asbestosis for example, the vast majority of occupational diseases are not uniquely "occupational." Even when a disease results from exposures found only in a working environment, the pathology produced is often indistinguishable from that which could occur from sources outside the workplace. The liver disease produced by chlorinated hydrocarbons, for example, may be indistinguishable from liver disease caused by alcohol consumption or even viral illness. Similarly, leukemia or aplastic anemia resulting from benzene exposure occurs in a background of naturally occurring leukemia and aplastic anemia. The lung cancer caused by coke oven emissions is indistinguishable from lung cancer caused by smoking, and even a case of industrial bronchitis is essentially indistinguishable from a case of bronchitis of other origin.

Moreover, some of the very hazards encountered at work may also be encountered outside the workplace. The stress-related diseases are a prime example. Even when it is undisputed that a particular stressor causes a particular disease, that noise causes hearing loss, for example, it is not uncommon to hear the argument that it was the noise from a loud stereo or from gunfire, rather than industrial noise which caused the hearing damage in a particular worker. In reference to psychological stressors, the problems are even of greater magnitude. Even when the relationship between emotional stress and cardiovascular disease is recognized, the contributory role of job-related stresses versus other life stresses is always contentious.

It must also be appreciated that the majority of chronic diseases are often multifactorial in origin, at least to some extent. For example, it is well known that an asbestos-worker who smokes has a much higher risk of lung cancer than an asbestos-worker who is a non-smoker, and that alcohol consumption increases the risk of liver disease in workers exposed to industrial liver toxins such as carbon tetrachloride. (Clearly those not exposed to asbestos have a lower risk than those who are exposed, and those not exposed to carbon tetrachloride have a lower incidence of liver disease than those who are exposed, regardless of their smoking habit or alcohol consumption. However, the non-occupational factors become confounders in the question of the occupational nature of these diseases.)

There is also much uncertainty as to how to classify the situation in which the health of a worker with a pre-existing disease is worsened by workplace exposures. For example, when a worker with pre-existing emphysema develops further breathing difficulties on exposure to dust or fumes in the workplace, there is controversy as to whether this is a case of "occupational" disease. There is also the ambiguity created when a non-occupational exposure aggravates a condition that originated at the workplace. For example, a heart attack brought on by jogging in a worker whose cardiovascular system was weakened by stress or even chemical toxins at the workplace may or may not be considered "occupational." The difficulties in determining the work-relatedness of diseases are discussed in detail by Peter Barth in Workers' Compensation and Work-Related Illnesses and Diseases[4].

3.1.2 *Health Impairment and the Definition of Disease*

The boundary between health and disease is not the clear-cut dichotomy it may seem to be. The World Health Organization (WHO) defines health impairment as a reduction in functional abilities and has set down criteria of health impairment (see Appendix 1). However, there is often vast disagreement among scientific authorities as to what constitutes impaired health. What is regarded as unacceptable is a matter of interpretation and differs significantly from country to country.[5] It should be appreciated that there is usually a continuum between health and disease: no observed effect -- homeostatic or adaptive effects -- early effect of debatable health significance -- early health impairment -- manifest disease. According to the WHO, there is a general agreement that, with regard to lead, for example, an aminolevulenic acid (ALA) level of 20 mg/litre in urine constitutes evidence of health impairment, although there is less agreement as to the relevance of a subclinical reduction in nerve conduction velocity, and no consensus regarding the relevance of change in hydroxyindoleacetic acid excretion. Similarly, with respect to noise, any overt dip in the audiogram is considered to constitute evidence of impairment, there is agreement as to the significance of temporary auditory threshold shift but no consensus regarding the relationship of noise exposure to presbycusis (impairment of hearing in old age).[6]

With the advent of increasingly sophisticated measures of health, deviations from normalcy can, for many diseases, be detected at early stages. (However, this does not always imply that the damage is reversible or even that the outcome can be altered, either by therapy or by removal of the worker from exposure). If disease is defined as any impairment in health, then the number of workers currently suffering from some form of occupational disease would be immense.

As will be illustrated below, the criteria used by the Workers' Compensation Board are much more rigorous. The significance of chest x-ray changes without abnormalities in lung function tests in workers exposed to silica, for example, has been the focus of considerable contention. (It is worth noting that the National Institute for Occupational Safety and Health -- NIOSH, the official research centre for occupational health in the U.S., includes these conditions as "occupational diseases.") Quite apart from the controversy as to the stage at which abnormal findings constitute disease, the debate rages on as to whether or not workers with detectable impairments, but no obvious "disability" should receive compensation.

While on the one hand some diseases are detectable well before they produce noticeable disability, on the other hand some occupational diseases cannot be easily documented and measured. These would include conditions characterized largely by subjective manifestations (symptoms), with few, if any, objective manifestations (signs). Headache and backache are common examples. Many workers may have been accused of malingering when in fact they may well have been suffering from an occupational disease. Diagnostic issues of this sort thus affect both the ability to adjudicate individual cases for purposes of compensation, and the assessment of the magnitude of the occupational disease problem.

3.1.3 *The State of Medical Knowledge, Exposure Records, and the Structure of Occupational Medical Services*

Aside from the etiological difficulties in defining the work-relatedness of a condition and the debate regarding the point at which an "impairment" becomes a "disease", a major obstacle in estimating the dimensions of occupational disease relates to the filtering role played by the attending physician. Occupational medicine still remains a miniscule part of the training of medical practitioners, and occupational medicine is still not a recognized specialty in Canada. Although there are a few diploma courses in occupational medicine offered, there is no financial incentive to entice medical practitioners to take these programs, neither OHIP nor the WCB grants preferential billing to those practitioners who have made the effort and financial sacrifice to acquire greater knowledge. Consequently, complete occupational histories are rarely taken by an attending physician and the possible work-relatedness of a disease is often never even contemplated. An article entitled "The Occupational History: A Neglected Area in the Clinical History" which recently appeared in a medical journal, noted the failure of monographic writings on physical diagnosis and family medicine to give emphasis to obtaining a substantial description of the patient's employment and its health effects. It was concluded that "no more attention is being given the patient's occupation today by the medical practitioner than it was three centuries past."[7]

A further contribution to this filtering process is the fact that usually the patient is unable to tell the doctor exactly what he or she works with. The Discher study, for example, found that a substantial number of workers did not respond to the survey question regarding past levels or types of hazardous exposures.[8] (See Appendix 2.) Even when the exposure is recalled, the composition of the substance is often kept secret. The National Occupational Hazard Survey conducted in the U.S. found that a great proportion of substances are known by trade name only and a large percent of these cannot easily be obtained due to the Trade Secrets Act.[9] An Ontario task force on occupational health found that even amongst the largest companies in the province, industrial exposure records are not kept.[10]

It must be appreciated that although Ontario's Designated Substances Regulations could result in a marked improvement in the compilation of exposure records, the scope of occupational disease far exceeds the number of hazards currently proposed for designation even including the few dozen on the horizon, and the process itself is exceedingly slow.

The result is that if an occupational exposure produces one of the so-called "ordinary diseases" of life, the lack of knowledge of the exposure will clearly prevent recognition and reporting of the disease as occupational. Even when the disease is essentially occupationally-specific, it may easily be misdiagnosed if occupational factors are not considered. Woodward and Fondiller found that physicians rarely correctly diagnose either beryllium or asbestos-related diseases unless the physician or the patient had prior knowledge of exposure.[11] According to Barth, type I errors in diagnosis, i.e. not attributing a work-related disease to the employment, are much more common than type II errors, i.e. falsely attributing a non-work-related illness to workplace factors,[12] resulting in a net under-recognition of occupational diseases.

Not only are there rather few physicians well trained in occupational medicine, but in Ontario, as elsewhere, the majority of occupational physicians are directly employed by management. Although the system of health care in Ontario is markedly different from that in the U.S., the organization of occupational medicine, based within the corporate structure, is remarkably similar. Davidson summarized labour's concerns regarding the company doctor as follows:

> The M.D. is in a peculiar position. If he tells a worker that his illness is job-caused, the employee may sue the company for Workmen's Compensation and subpoena the doctor to testify. At best, the typical medical man dislikes appearing

> on a witness stand. It must be even more difficult when he is put in a position of testifying against the company which has paid his fee . . .[13]

Compensation battles in Ontario do not take place in a courtroom. Nonetheless, even filing a compensation claim for a patient may pose the same sort of conflict. Moreover, the obligation of the physician to the company often leads to a predominantly palliative rather than preventive approach to occupational disease in which the emphasis is on screening the individual worker rather than the work environment. A prominent occupational health text book states:

> The pre-employment medical examination provides an opportunity to exclude persons who are likely to be susceptible to particular hazards. For example, those who have a history or other evidence of chronic respiratory disease or disability should not be employed in occupations which involve exposure to dusts or other aerosols which are likely to exacerbate their condition. Persons with a history of atopy or chronic skin disease should as far as possible be excluded from work which exposes them to irritants or allergens. A history of liver disease . . . would be a good reason for exclusion from a job which entails exposure to liver poisons . . .[14]

This approach of continually but exclusively exposing "healthy" workers to known hazards has been vehemently opposed by labour organizations. Objecting to the notion that only certain workers are susceptible to these hazards, they have dubbed this practice the "hypersusceptible worker syndrome" and criticized it as discriminatory. In that the occupational physician plays a key role in this approach, the company doctor has become the focus of criticism.[15] This, in turn, may erode the doctor-patient relationship and may contribute to the failure to recognize new cases of occupational disease.

Thus, not only the lack of training in occupational medicine but also the structure of occupational medicine in Ontario militates against the full recognition of occupational disease.

3.1.4 *The State of Scientific Research*

Although lack of knowledge by the attending physician is a significant factor limiting the recognition and reporting of occupational disease, an even greater obstacle, preventing even the most well-informed and well-intentioned practitioner from accurately assessing the work-relatedness of a disease, is the dearth of scientific research in the area. Threshold limit values exist for only 400 of the 12,000 or so identified toxic materials and few of these have been adequately studied. Yet countless new chemicals and processes are introduced into industry annually with minimal if any pre-testing as to their toxicity. It is indicated that there are over two million chemical compounds in use.[16]

Scientific research is also subject to the influence of limitations in the training and organization of occupational medicine, as well as to the labour-management conflict. The preliminary decisions by which research is initiated, the decisions that govern priorities in the allocation of resources for research and the decisions relating to the circulation and use of research results are all subject to political pressure. The potential for conflicting interests manifesting themselves in the actual methodology is also of relevance. As Ison notes:

> Where conflicts of interest are involved, the efficiency of peer-group review can surely be impaired if the opportunity and ability to question the research are unevenly distributed. Some research in occupational health is done by doctors employed in universities, or in foundations of a semi-independent nature. Some research is done by doctors employed directly or indirectly by industrial corporations. But workers' organizations have not had comparable resources, and very little medical research has been done by doctors employed by unions. The research work of doctors employed at universities and elsewhere is subject to the critical screening of doctors employed by major corporations. But it is not usually subject to scrutiny by doctors employed by workers.[17]

Limitations in occupational health research clearly hinder the full appreciation of the dimensions of occupational diseases.

3.1.5 *The Changing Incidence of Occupational Diseases*

The etiological and diagnostic issues discussed above, the lack of agreement as to how to define occupational diseases and the state of medical knowledge and research are all obstacles to estimating the dimensions of occupational disease. A further, perhaps even more important fact, is that the character and incidence of various occupational diseases are ever-changing.

The rapid proliferation of new chemicals and processes is cause for concern, particularly considering that many occupational diseases have a latent period of years or decades. Hence, the ill-effects of these often completely untested new chemicals can only be speculated upon.

The increasing use of modern technology in the workplace has created a great many new potential hazards. Recently, the introduction into the workplace of video display terminals (VDTs) has sparked public concern and several incidents involving birth defects potentially linked to VDTs have led to a government inquiry into their safety.

The increasing number of women in the labour force generally and particularly in "non-traditional" jobs, has focused greater attention on reproductive health hazards. NIOSH has already listed twenty chemicals as potential sources of birth defects and miscarriages. It should be noted that although there are substances that are particularly hazardous to the fetus when a woman is exposed during pregnancy (teratogens), other substances (mutagens) can cause chromosomal abnormalities in both sexes. The film The Song of the Canary, for example, focused public attention on the tragedy of DBCP (dibromochloropropane) which caused infertility, among other things, in the men employed at the chemical plant that produced the chemical.

The changing composition of the labour force leads to changes in occupational disease patterns. For example, there has been a decline in the number of agricultural workers and an increase in the industrial work force generally, with increases in some sectors and decreases in others.[18] Chapter III will illustrate, for example, how the marked changes in the size of the work force employed in uranium mines affected the incidence of silicosis in Ontario.

Barth enumerated other factors that may affect the prevalence of occupational diseases. He surmised that the increased role of regulatory authorities, the increasing importance of health and safety issues in collective bargaining, the development of technological improvements in monitoring and controlling hazards likely all have had some beneficial impact on the work environment. He also concluded that workers' compensation laws have had an influence on working conditions and have thereby served to reduce the incidence of disease.[19]

Many changes in disease incidence attributable to a reduction in exposure to the hazard have been documented. For example, the incidence of beryllium-related disease has been substantially reduced due to the elimination of its use in 1949 by the three major fluorescent lamp manufacturers in the U.S.[20] Countless examples have, in fact, been recorded, attributable to substitution of a known hazard by a less toxic equivalent or to technological improvements in workplace design and industrial hygiene practice. However, although scientific knowledge concerning the hazard, and technological advances in the means of control are important variables in the incidence of disease, Barth concludes that economic factors are the crucial variables determining the incidence of occupational diseases.[21]

It is difficult to speculate to what extent the increasing number of hazards in the workplace is being balanced by efforts to improve working conditions. In any case, the dimensions of the occupational disease problem are of no small concern and all indications are that it will continue to be so for years to come.

3.2 *Estimates of the Dimensions of Occupational Disease*

There are no published estimates of the incidence and severity of occupational diseases in the province of Ontario. Given the embryonic nature of available data, and considering the issues enumerated above, it is not surprising that this mammoth and complex endeavour has not been undertaken here. It is useful, nonetheless, to examine the estimates that have been made elsewhere. Although the crude nature of these estimates must be fully appreciated, some very rough conclusions can be drawn and some extrapolations can be made to the situation in Ontario. According to the requirements set out in the Black Lung Benefits Reform Act of 1977, the U.S. Secretary of Labor, in co-operation with NIOSH, conducted a study to assess the magnitude and severity of occupational disease, the status and adequacy of current disability benefit programs for victims of occupational diseases, and the status and adequacy of federal health and safety programs.[22] The study included several research projects conducted by noted experts in occupational medicine, as well as various short-term analytical studies conducted by the project staff.

3.2.1 Data Based on Exposure Estimates

First, an analysis was undertaken of the epidemiological statistics on ten well-documented hazardous substances which caused chronic respiratory disease or cancer of the respiratory tract. An estimate was made of the excess disability and/or premature death attributable to exposure to asbestos, cotton dust, silica, beryllium, cadmium, arsenic, chromium, nickel, coal tar products and isocyanates. Data on the number of workers exposed were taken from the Bureau of Labor Statistics and from the National Occupational Hazard Survey (NOHS) conducted by NIOSH. It was felt that the number of exposed workers is generally undercounted due largely to worker turnover. Other limitations of the data enumerated were related to the inadequacy of available data on exposure intensity and duration, the absence of information on possible confounding exposures and the limitations in both number and quality of the epidemiological studies.

As data even on the number of workers exposed to various hazards are unavailable in Ontario, no attempt can be made to apply this technique to the Ontario situation. In 1975, the Interministerial Task Force on Occupational and Environmental Health, established a list of 18 significant known toxic agents, substances or conditions of immediate concern, together with an approximate number of employers and employees under investigation by the Ministry of Health (see Appendix 3). It was stressed that the people exposed in Ontario to these known agents are really very many more than the 68,000 or so listed. The report states: "For example, although 64 employers (4,587 employees) are currently under investigation for noise exposure, some 500,000 employees in Ontario are probably exposed to noise at the 90 decibel level."[23]

3.2.2 Employer Records, Physician Reports and WCB Data

As the limited availability of epidemiological research was clearly unable to provide a broad picture of work-related disease, this Department of Labor (DOL) study examined other data sources, including employer records required by OSHA, state workers' compensation records and physician reports. It was found, however, that employer records understated the occupational disease problem largely because given the long latency period, affected workers are often no longer employed by the firm where the exposure occurred. It was also felt that employers were reluctant to report occupational diseases, even when they were diagnosed, because of their potential financial liability. Discher found that these logs very substantially understate all occupational disease problems, not merely those difficult to diagnose. (See Appendix 2, Table B.)[24] The situation in Ontario with respect to employer obligations to report occupational disease is alluded to in following chapters; data from the survey conducted for this Inquiry found that 15-20% of serious industrial disease claims are actually "initiated" by the employer. However, no systematic employer reporting has occurred in Ontario.

Physician-based records as exist in California were also examined by the DOL. Data from the Doctor's First Reports indicated that there were 5.1 occupational disease cases per 1,000 employees and 1.3 per 1,000 who suffered from an occupational disease that resulted in some lost work time. It was noted, however, that 97.2% of the cases recorded in the Doctor's First Report did not involve hospitalization. The DOL concluded that this source also understated the occupational disease problem because few physicians are trained to recognize chronic occupational diseases and cancer. Physician-based records in reference to work-relatedness of disease also do not exist in Ontario.

The DOL report concluded that workers' compensation data also understate the problem of chronic occupational conditions and cancer. The cases reported were markedly skewed to those easily recognized as work-related such as skin diseases, inflammations and poisoning. Part B of this chapter illustrates that this is also true in Ontario.

3.2.3 Death Certificate and Social Security Data; Mortality by Occupation and Attributability Estimates

Death certificate data were considered by the DOL project staff, however, they felt that various difficulties precluded their usefulness in estimating the magnitude of occupational diseases. Several studies comparing diagnostic evaluations with cause of death as listed on death records have found the latter to be grossly inaccurate. Other problems included the nonstandardization of occupational entries and difficulties in linking death certificates with study populations. (The standardization problem characterizes the situation in Ontario as well.) Current efforts by Statistics Canada to improve record linkage are described in the next chapter.

Social Security Administration data on medical diagnoses, demographic characteristics, occupation, and industry for disabled individuals were also examined by the DOL project staff. Although the data suggested large interoccupational and interindustrial differences in disease, the authors concluded that given the long latency periods in certain diseases and the extent of worker turnover, using this technique to estimate the incidence of occupational disease may be misleading. Insufficient data are available from the Canada Pension Plan to even contemplate using this technique here.

It should be noted that the often quoted U.S. figure of 100,000 annual deaths from occupational disease was derived from applying the "excess mortality" method. This is based on the eminently reasonable premise that in occupational groups whose mortality rates are greater than the general population (adjusting for age and social class), the excess death rate is attributable to the workplace. However, as the death registries existing in the U.S. lacked comprehensive occupational information, it was necessary to resort to the data provided in the 1951 Registrar-General's Occupational Mortality Report for England and Wales. (This is still considered the only sufficiently detailed source of mortality information by occupational group.) The transferability of these possibly out-dated figures is certainly open to question. (In attempting to answer the question of how much mortality of an occupational group is attributable to the work environment, Fox and Adelstein recently analyzed the 1970-72 Registrar-General's Occupational Mortality report. They concluded that 18% of the variation in mortality rates between occupational groups was workplace-related and 80% was related to "social class."[25] The distinction between these two categories; however, is certainly not clear-cut.)

An even cruder method of estimating the number of deaths from occupational disease is derived by allotting an estimate of the environmental sources of the major causes of death to the numbers of people dying from each of these causes. In Barth's illustration of this method he allots 5-10% of cardiovascular deaths as occupational, 15-25% of cancer deaths and 10% of pulmonary deaths. Applying these figures to the data on cause of death in Ontario (see vital statistics reprinted in Appendix I of Chapter I, Part B), would mean that 1,105 to 2,215 deaths from occupationally-related cardiovascular disease, 2,038 to 3,396 deaths from occupational cancer and 283 deaths from work-related lung disease occur annually in Ontario, for a possible total of almost 6,000 occupational deaths from these three categories of disease alone. Clearly, the quality of this estimate is dependent on the validity of the estimated percentage, which in turn is dependent upon a number of complex factors again including the definition of "occupational" utilized. Nonetheless, these figures at least provide the impression that workplace factors may be contributing significantly to deaths in this province.

3.2.4 *Self-Reporting of the Work-Relatedness of Disease*

Because the traditional data sources (particularly those discussed in Section 3.2.2) underestimate the magnitude of the occupational disease problem, an interview survey was undertaken by the U.S. DOL of 18,000 disabled and nondisabled adults aged 20-64. (As the data do not include individuals over age 65 or those who have died from occupational diseases, occupational cancer was omitted from these data.) The results indicated that there were almost two million workers in the U.S. disabled by occupational disease. Approximately 700 thousand were suffering from long-term total disability and another 1.2

million were partially disabled. The major job-related disabling conditions reported by workers included back and spinal conditions (34%), heart and circulatory illnesses (26%), respiratory conditions (13%), mental illnesses (9%) and digestive conditions (8%).[26]

This approach is of particular interest in that the information was based on the assertion that an individual with a diagnosed medical condition is able to determine the work-relatedness of that condition. There was no direct link to compensation benefits, hence there was no financial incentive for individuals to overstate the occupational origin of their illness. As this approach has been seldom used, the data were checked for consistency both internally and externally.

The results showed that about 17% of the disabled population believed their disability to be work-related -- 5% from diseases resulting from job injuries and 12% from occupational diseases. When those disabled and nondisabled individuals polled in 1972 were recontacted in 1974, the figures remained relatively constant. Further, nine major health conditions reported in the survey were examined separately and again a similar pattern of response was demonstrated (see Table A in Appendix 4).

To further evaluate the appropriateness of individual responses, types of diseases were examined by occupational group. Again, a consistency was demonstrated -- for example, those in occupational categories with the heaviest exposure to dusts and fumes did indeed show the overwhelming majority of occupationally-related respiratory cases (78%). Similarly, 73% of the cases of occupational cardiovascular disease occurred in those classes of workers felt by the authors to be subject to the highest stress levels based on the available knowledge of the work-relatedness of cardiovascular disease (see Table B in Appendix 4). Also, analysis was undertaken of exposure to five physical and chemical hazards known to be associated with respiratory disease (dust, fumes, heat, cold and dampness). The study found that of those workers with severely disabling respiratory disease who were exposed to the adverse workplace conditions listed, 23.4% cited a job-cause, whereas of those with severe respiratory disease but who were not exposed to fumes, dust or smoke, only 2.4% thought their condition was work-related. It was therefore concluded that individual judgment or subjective impressions can provide reasonable indicators of job-relatedness. The authors point out in fact that self-reporting would tend to underestimate diseases where the etiology is unknown or complex, where the disease progresses insidiously, where there is long latency, or where the disease is related to low level exposure. Other authors have alluded to the erroneous tendency to underrate the worker's own knowledge of his or her working conditions and state of health,[27] and studies elsewhere have demonstrated the validity of workers' judgments about the environment and health conditions.[28]

Not surprisingly the DOL has come under fire from the National Council on Compensation Insurance for its use of this survey.

They point out that the survey may have included a large number of people who were disabled from work accidents in their count of those with occupational diseases; they criticize the DOL for not reporting its sampling errors; they note that less than a thousand people were actually interviewed and that the survey was eight years old; and they assert that the large number of job-related questions may easily have led to biased estimates of the occupational factors.[29] Despite these flaws, the magnitude of the occupational disease problem revealed by the report cannot be completely discounted.

4. The Issues Summarized

1. The scope of occupational diseases is extremely vast. Physical agents, biological agents, chemical agents, biomechanical factors and psychosocial characteristics associated with various occupations or found in various workplaces, may cause or contribute to adverse health in a multitude of different ways depending on a wide variety of circumstances.

2. There is little consensus as to how large a role the workplace must play in the causation of ill-health in order for the adverse condition to be considered of "occupational" origin. There is also little consensus as to how much impairment must be present for an adverse condition to be considered a "disease." Hence the magnitude of the occupational disease problem is difficult to quantify. The inadequate knowledge about occupational health in the medical community, the lack of exposure records and the corporate basis of occupational medical services add to the difficulties in this area. Moreover, scientific research on occupational diseases is sorely lacking. Further, as the nature of modern technology and industrial processes change, the nature and extent of exposure to various hazards changes, and accordingly the character of occupational diseases also changes.

3. Despite these difficulties, various attempts have been made to estimate the role of the workplace in the ill-health of society. The different sources of data all contribute to some extent, but major methodological difficulties characterize all approaches. Nonetheless, it seems that the extent of occupational diseases far exceeds that reflected in workers' compensation statistics. The conclusion reached by the U.S. Department of Labor is that victims of disease are not well-served by the American workers' compensation system. Concern has been expressed that some of the factors contributing to this failure may indeed be applicable to Ontario as well.

With this as background, the variety of approaches to compensating occupational diseases in various jurisdictions will be reviewed in Part B of this chapter and the Ontario experience critically examined.

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APPENDIX 1

Criteria of Health Impairment

Implications for Workers' Compensation of the Early Detection of Health Impairment

The following is an excerpt from a report of the World Health Organization entitled -- "Early Detection of Health Impairment in Occupational Exposure to Health Hazards." As the purpose of health impairment criteria is to prevent manifest disease, it is assumed that the detection of these changes will trigger some intervention. There is no clear consensus in Ontario as to the criteria for removal of a worker from exposure while a problem is being investigated or rectified. Nevertheless, the principles of workers' compensation imply that an affected worker will not suffer any financial loss as the result of a health impairment having been detected. Workers' compensation may reasonably be expected therefore, to provide compensation to a worker in the early stages of impairment.

Excerpt

Criteria of health impairment should, if possible, be based upon early reversible changes that predict the occurrence of manifest signs and symptoms, in order to make prevention of overt disease or disablement possible. One may distinguish the following broad overlapping categories of criteria:

1. Changes in biochemical and morphological parameters, to be measured by laboratory analysis, e.g. parameters of disturbed porphyrin and glutathione levels in exposure to lead; inhibited cholinesterase activity in exposure to organophosphorus insecticides; changes in the levels of various serum enzymes; changes in metal concentrations in body fluids; chromosome aberrations; or abnormal sputum cytology. Some changes are detectable only after loading tests, e.g. biliary excretion of dyes, or by other special tests.

2. Changes in the physical state and the function of physiological systems, to be evaluated by physical examination, and by means of loading tests, e.g. pallor, stooping posture, and changes in ECG tracings, in physical working capacity, in higher nervous function, or in reproductive function.

3. Changes in well-being to be evaluated by medical history taking and questionnaires, e.g. drowsiness, irritation of mucosa on exposure to solvents.

4. Integrative changes that may result from effects on several physiological systems: more so than the previously listed changes, they are not specific and are affected by factors unrelated to work, e.g. nutrition, communicable diseases. However, they may be important in health evaluation for elucidating work-related health effects and may at times be the only available evidence of an effect. In individual cases, it may not be possible to substantiate their relationship to work factors unless, for example, the time course of events in relation to exposure is highly suggestive. However, in epidemiological surveys with adequate study design, these integrative changes may indicate the beginning of health impairment due to work factors.

   Examples are changes in:

   - subjective state, the incidence and prevalence of which can be measured objectively in groups of workers (often the help of psychologists or sociologists is needed to prepare adequate questionnaires);

- behaviour, e.g. excitation, drowsiness, depression, personality changes;
- anthropometric measures, e.g. height, weight, girth;
- employee records, e.g. accidents, sickness, absenteeism;
- medical statistics; e.g. hospital admission rates, mortality rates.

These integrative changes may often be the first indication of hitherto unexpected causes of ill-health that require more detailed observation. In addition to being potential indicators of work-related factors, they may also be useful in providing insight into the general health of the working population as determined by the general environmental or socioeconomic conditions.

APPENDIX 2

"Occupational Disease is Grossly Under-Reported": A Look at the Discher Study[1]

This pilot study to determine the usefulness of a survey method for obtaining data on occupational disease was conducted for NIOSH from July, 1972 through August, 1974. The study had two specific goals:

1. To determine the utility of procedures designed for finding occupational disease in a cross-sectional survey; and,

2. To ascertain how much new data could be generated by this method.

The overall sample included 140 establishments from 3 selected urban areas and 1 selected agricultural area. The selection from the urban areas was purposely designed "to assure that the results would yield cases of ocupational disease of special interest to NIOSH." The sample was stratified according to target groups: lead-using industries, chlorinated hydrocarbon solvent-using industries, and agriculture. All other hazardous industrial categories in the sample formed a fourth stratum called "other non-agricultural industries," which was under-sampled compared to the lead and chlorinated hydrocarbon strata.

All the establishments were given 10 to 20 days notice of the industrial hygienist's visit, and the firms that refused to participate in either the walk-through survey or the subsequent medical survey were allowed to drop out of the sample. The hygienist's environment survey involved solely the use of "subjective judgment" regarding the exposure of workers to all hazards. The sole exception to this was the noise hazard, where test instruments were used. After the walk-through by the hygienist, a limited number of these plants were selected, and only production workers in these establishments were asked to participate in the health survey. "The plants included in the medical survey were those in which the workers were exposed to the target hazards and/or other hazards inadequately controlled as determined by the industrial hygienist."

The medical survey was carried out on 1,400 employees by a team consisting of a physician, nurse, and para-medical person. Aside from some tests given to all survey participants, in many of the examinations checks were made tailored to the worker's specific exposure.

Over 1,100 medical conditions were found among 908 ultimate participants. As shown in Table A, it was found that those which were probably occupational constituted 31%; probably non-occupational, 45%; doubtfully occupational or can't evaluate, 14%; and suggestive history, 10%.

Of 346 cases of probable occupational disease, hearing loss was most frequent, 28%; then skin, 18%; lower respiratory conditions, 14%; toxic and low grade toxic effects and non-symptomatic conditions (mainly elevated blood levels), 14%; upper respiratory conditions, 11%; and eye conditions, 9%. Anemia, disease of the musculoskeletal and connective tissues and other conditions accounted for the remaining 6%.

Those known to have been exposed to poorly controlled hazards had a higher prevalence rate (39%). Of the 908 workers participating in the medical survey, 258 workers were found with 346 cases of probable occupational disease, for a prevalence rate of 28.4% per 100 workers. Occupational exposures were determined for all workers in the survey.

As noted by Barth,[2] "with the sampling procedures used by the group, it is not surprising that significant exposures to hazardous substances and cases of occupationally-related impairments were found. However, such estimates were based on a very small, non-random sample and the procedures used to identify diseases were limited, thereby undermining general applicability of the findings to all diseases found or accounts of all individual disease cases." Discher and his associates recognized the biases within their reported data because of the highly limited and one-time-only medical examination. They pointed out that bias in the study tended towards under-recognition of probable occupational disease, ranging from missed diagnosis due to the omission of many procedures, to ascribing the relationship of the disease to the doubtful or non-occupational instead of probably occupational category because of missing data. Thus, no quantitative measures of the disease problem should be drawn from the study, but the conclusion by the authors that "a vast reservoir of unreported occupational diseases exists in U.S. industry," is still a valid one.

Discher and his associates strongly recommended that a nation-wide survey be carried out based on the techniques developed in this pilot study, for an estimate of the extent and kind of occupational disease. Unfortunately, this never did take place.

Many of Discher's other findings were also significant, for example, the fact that workers did not know or could not recall their past exposures to hazardous substances: for 17 out of 27 questions relating to exposure, over 20% of the participants left blanks, which were then classified by the researchers as unknown.

Also of interest, were the findings when the survey morbidity data were checked for duplication with records of compensation claims; and injury and illness records which employers are now required to keep under OSHA regulations (Employer's Log). As shown in Table B, of 451 reports of occupational disease, 89% were found on the survey alone; 2% on the employer's log; and 3% on compensation claims only; 6% were duplications. The lack of compensation claims for probable occupational diseases is of considerable interest.

Notes

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Table A

Medical Condition by Occupational Relationship

Medical Condition[1]	Total	Probable Occupational Disease	Can't Evaluate and Doubtful	Suggestive History	Probable Non-Occupational Disease
Total	**1,116**	**346**	**151**	**113**	**506**
Anemia and Other Diseases of the Blood	49	7	21	1	20
Conjunctivitis and Other Conditions of the Eye	74	31	1	24	18
Refractive Errors	84	0	1	0	83
Hearing Loss, mild	86	68	12	0	6
Hearing Loss, moderate	32	26	4	0	2
Hearing Loss, advanced	4	2	2	0	0
Hearing Loss, possible threshold	16	0	16	0	0
Hypertension	138	1	0	0	137
Heart Disease	14	0	1	0	13
Other Diseases of the Circulatory System	3	0	0	0	3
Respiratory Conditions, excluding upper	289	49	76	11	153
Upper Respiratory Conditions	73	38	2	21	12
Skin Conditions	128	64	4	35	25
Diseases of Musculoskeletal and Connective Tissues	28	9	2	7	10
Symptoms and Ill Defined Conditions	17	1	6	1	9
Non Symptomatic Conditions	27	27	0	0	0
Toxic and Other Adverse Effects	23	9	1	13	0
Low Grade Toxic Effects	14	13	1	0	0
Other Conditions	17	1	1	0	15

Notes: Probable occupational disease - Manifestations of disease are consistent with those known to result from excessive exposure to a given injurious agent; this injurious agent is present in the patient's working environment and significant contact in course of usual duties is likely.

Doubtful occupational disease - 1) Manifestations of disease are not entirely consistent with those known to result from excessive exposure to an injurious agent or 2) manifestations of illness are consistent with those known but no significant contact with alleged injurious agent can be established.

Suggestive History - Manifestations of disease as given in history are consistent with those known to result from excessive exposure to a given injurious agent; this exposure is occurring or has taken place; there is no objective evidence of disease at the time of the medical examination.

Cannot be evaluated - An association between disease and occupational factors has never been clearly established; e.g. cardiovascular disease, respiratory infections, mental and psychoneurotic episodes.

Non-occupational - Manifestations of disease but no relationship to occupational exposures.

Source: Occupational Disease Surveillance Study, University of Washington, Department of Environmental Health.

Table B

Comparison of Probable Occupational Disease Conditions Found on Medical Survey with the Employer's Log and Workmen's Compensation Claims

Probable Occupational Disease Condition	Total	Survey Only	Log Only	Compensation Claims Only	Survey and Log	Survey and Claims	Log and Claims	All Three
Total	**451**	**399**	**11**	**13**	**2**		**20**	**6**
Anemia and Other Diseases of the Blood	7	7						
Conjunctivitis and other Conditions of the Eye	40	30		2	1		7	
Refractive Errors								
Hearing Loss, mild	113							
Hearing Loss, moderate	39	39						
Hearing Loss, advanced	4	4						
Unknown Hearing Loss	1			1				
Hypertension	1	1						
Respiratory Conditions excluding upper	51	49	1				1	
Upper Respiratory Conditions	39	39						
Skin Conditions	76	58	6	3	1		3	5
Disease of Musculoskeletal and Connective Tissues	13	8		2			2	1
Symptoms and Ill-Defined Conditions	1	1						
Non-Symptomatic Conditions	27	27						
Chemical Burns	6		1	3			2	
Toxic and Other Adverse Effects	17	9	3	2			3	
Low Grade Toxic Effects	13	13						
Other Conditions[1]	3	1					2	

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[1] Includes conditions such as diseases of digestive system and epididymitis.

Source: Occupational Disease Surveillance Study, University of Washington, Department of Environmental Health.

APPENDIX 3

DATA ABOUT EXPOSURE IN ONTARIO
Excerpt from the Ontario Interministerial Task Force on Occupational and Environmental Health, 1975*

Significant Known Toxic Agents, Substances or Conditions

A tentative list of significant known toxic agents, substances or conditions of immediate concern are shown below together with approximate numbers of employers and employees currently being investigated by the Ministry of Health.

Significant Toxic Agent, Substance or Condition	**Number of Employers**		**Number of Employees**	
Arsenic	4		234	
Asbestos	100		3,500	
Benzol	1		49	
Beryllium	4		110	
Carcinogen - Benzophyrene	4		-	
Cotton and other vegetable fibers	5		1,600	
Fluorides	4		537	
Hard Metals (cobalt)	11		260	
Ionizing Radiation	1,000		20,000	
Isocyanates	38		1,300	
Lead	100		5,500	
Mercury	17		900	
Nickel Carbonyl	1		7	
Noise	64		4,587	
Organophosphorus	10		520	
Silica	164	+ mines	22,133	R
			6,710	NR
Talc	1	mine	578	
	15	other		
Vinyl Chloride	340	PVC	?	
	8	VCM	?	
Total	**1,891**	**+ mines**	**68,525**	

The above figures are for companies brought to the attention of the Ministry of Health and currently under investigation by it. The number of persons exposed in Ontario to these significant known toxic agents would be very much greater, however. For example, although 64 employers (4,587 employees) are currently under investigation for noise exposure, some 500,000 employees in Ontario are probably exposed to noise at the 90 decibel level.

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* This excerpt is included here as a rough indication of the extent of knowledge by the Ontario Government as to exposure to toxic agents prior to the establishment of the Occupational Health Branch in the Ministry of Labour. Clearly, knowledge on the extent of exposure has improved considerably over the last seven years; the Annual Reports of the Ministry of Labour list the number of plants investigated, the number of biological samples analyzed and the number of workers otherwise monitored. However, no updated figures on the total number of workers exposed to various hazards were obtainable.

APPENDIX 4

Results from Survey of Disabled and Nondisabled Adults[1]

Table A

Percentage of Disabilities Related to Occupational Diseases[2]

Health Condition	Number Disabled from All Causes[3]	Percent Related to Occupational Disease
All	14,316	11.6
Musculoskeletal	5,244	10.7
Cardiovascular	3,084	13.7
Mental	1,273	11.6
Respiratory	1,371	16.1
Digestive	704	18.3
Neoplastic (cancer)	335	1.5
Neurological	252	3.6
Endocrine	275	1.5
Urogenital	289	2.1
Other	1,489	7.9

Table B

Occupational and Disabling Health Conditions for Occupational Disease Cases (in thousands)[1,2,3]

Disease	Pro-Tech-Mgr-Adm.	Sales-Clerical	Crafts-Operatives	Labourers	Service Workers	Other	Total
Musculoskeletal	9 (3.1%)	25 (8.5%)	163 (55.4%)	30 (10.2%)	42 (14.3%)	24 (8.2%)	294
Cardiovascular	71 (19.8%)	49 (13.7%)	87 (24.3%)	38 (10.6%)	104 (29.1%)	9 (2.5%)	358
Respiratory	18 (11.5%)	9 (5.8%)	100 (64.1%)	22 (14.1%)	4 (2.6%)	4 (2.6%)	156
Digestive	10 (10.3%)	21 (21.6%)	28 (28.9%)	15 (15.5%)	24 (24.7%)	1 (1.0%)	97
Mental	12 (8.8%)	22 (11.2%)	38 (27.9%)	4 (2.9%)	57 (41.9%)	3 (2.2%)	136
Other	- (0.0%)	1 (4.8%)	8 (38.1%)	4 (19.0%)	4 (19.0%)	5 (23.8%)	21
Total	**120 (11.3%)**	**126 (11.9%)**	**424 (39.9%)**	**113 (10.6%)**	**234 (22.0%)**	**46 (4.3%)**	**1,063**

Notes

1. Source: 1972 and 1974 Survey of Disabled and Nondisabled Adults Ages 20-64 in 1972 and 22-63 in 1974. SSA/HEW. Analysis and tabulations prepared by ASPER, U.S. DOL, August 1979.
2. These results were obtained from the 1972 Survey. Followup in 1974 produced remarkably similar findings (e.g. total % related to occupational diseases was 11.3%).
3. Includes severe as well as partial disability.

B: COMPENSATING OCCUPATIONAL DISEASES

1. Facts and Figures

Ontario

The number of diseases compensated by the Ontario WCB is small in comparison to the number of accidents. In 1980, 411,476 claims were allowed, of which 7,611 or only 1.8% were for occupational diseases. This has consistently been the case for at least the last five years, as shown in Table IB-1. However, industrial disease claims tend to be more serious than accident claims. Whereas about 35-40% of total claims are associated with disability or lost time, more than 75% of disease claims require payment of compensation benefits. Nonetheless, the proportion of compensation benefits attributed to disease is still less than 4% of total compensation benefits awarded. Moreover, as shown in Table IB-2, the vast majority of occupational disease claims involve short-term or easily recognizable conditions, such as burns (accounting for almost 30% of the claims), acute poisoning (24%), hearing loss (18%), skin diseases (10%) and inflammation of joints, tendons, muscles or arteries (8%).[1]

Although some of these conditions, occupational deafness for example, can have a permanently devastating effect on the quality of life of the afflicted individual, the percentage of disability award, according to the claims survey conducted for this Inquiry, ranged from 2% to 13% and averaged only 5.2%.[2] Though there is certainly room for debate as to the appropriateness of these disability assessments, it is clear that noise-induced hearing loss is not treated as a particularly disabling disease according to the current compensation policy.

What is certainly evident from Table IB-2 is that the serious chronic diseases such as cancer, heart disease and respiratory disease which account for the bulk of mortality in the Ontario population[3] (see Table A in Appendix 1), as well as a great deal of morbidity[4] (see Table B in Appendix 1) constitute only 2.7% of the industrial disease claims or less than .05% of total claims allowed. This is in marked contrast to Canada Pension Plan awards in which 95% of the total benefits are for diseases.[5]

When permanent disability benefits are granted for these diseases, their increased seriousness is indeed reflected by the higher assessments awarded. Although 88% of the allowed cancer claims in our survey were death claims, the permanent disability benefits that were received ranged from 40% to 100% awards, averaging 69%. The permanent disability awards received for silicosis averaged 23%.[6] Thus, the more costly industrial disease claims are very few in number.

Other Canadian Jurisdictions

The experience in Ontario is similar to that in other jurisdictions where occupational disease claims account for but a small percentage of total claims and these consist largely of acute and/or easily recognizable (and less costly) conditions. In Quebec, about 2.7% of their 195,000 newly diagnosed claims in 1979 involved industrial diseases.[7] Of the 6,552 industrial disease claims received in 1980, 97 respiratory disease claims were allowed as were 31 cancer claims.[8] In British Columbia, the 4,212 industrial disease claims allowed in 1980 represented 2.6% of their 159,994 total allowed claims.

Table IB-1

Total Allowed Claims and Allowed Industrial Disease Claims, 1976 - 1980

Year	Total Claims[a]	Compensation Claims	%	Industrial Disease Claims[b]	Industrial Disease Compensation Claims[c]	%[d]	Total Industrial Disease Claims as % of Total Claims	Industrial Disease Compensation Claims as % of Total Compensation Claims
1980	411,476	165,221	40.1	7,611	5,905	77.6	1.8	3.6
1979	413,008	165,456	40.1	7,829	5,899	75.3	1.9	3.6
1978	385,945	153,085	39.7	6,834	5,702	83.4	1.8	3.7
1977	395,146	150,904	38.1	7,691	6,015	78.2	1.5	3.9
1976	433,799	154,520	35.6	6,310	5,766	91.3	1.4	3.7

Notes

(a) Total claims include compensation claims as well as "medical aid only" claims.

(b) Statistics on total industrial disease claims are maintained by Dr. Dorothy Burton of the Medical Division.

(c) Statistics on compensation claims are maintained by the Program Planning and Statistical Services Branch of the WCB.

(d) As b and c are from different sources, these percentages should be regarded as rough estimates.

Among these were only two cancer claims, 17 cardiac claims and 28 respiratory disease claims as shown in Table IB-3, amounting to barely more than 1% of the disease claims and .03% of total claims[9]-even less than the comparable figures for Ontario.

The other Canadian jurisdictions which responded to the questionnaire sent by this Inquiry illustrated a similar pattern. Saskatchewan allowed 218 claims for industrial disease in 1980, including two cardiac claims and six claims for respiratory disease.[10] Of the 916 industrial disease claims allowed in Alberta in 1980, 12 claims were for respiratory diseases and 27 were cardiac claims, again accounting for less than 5% of their total industrial disease claims. It should be recognized that definitions of "disease" may differ in the various jurisdictions and therefore the figures are not always exactly comparable. It seems, for example, that in Alberta where chemical burns involving eyes or skin were unlikely to be included in their listings of industrial diseases, systemic poisonings affecting the respiratory system accounted for 657 or 72% of the allowed claims, poisoning affecting other body systems accounted for 131 or 14%, and noise-induced hearing loss accounted for 78 or another 8.5%, leaving only 0.5% unaccounted for.[11]

The U.S.

Barth found that the most common occupational disease, accounting for at least one-third of all cases in the United States, involved some type of dermatological disorder. He found that the inflammatory diseases of joints, muscles and so on, as well as systemic poisonings were also common, in keeping with the pattern shown in Canada. Respiratory diseases seemed to constitute a greater percentage of diseases compensated in the U.S. as compared to Canada; Barth noted that lung diseases were the most common occupational disease in Ohio, clearly attributable to the coal mining industry and Black Lung legislation. He also pointed out that heart cases are fairly common in some jurisdictions.[12] (Several tables from Barth's study are reproduced in Appendix 2.)

Europe

In Europe, the pattern was also similar. In England, occupational disease claims accounted for 2.3% of injuries due to accidents. Again, the largest number of diseases involved dermatitis, about one-third of cases involved inflammation of the joints or tendons, and only a trivial number involved serious diseases such as cancer. (See Appendix 2.) The data provided for France and for Denmark also showed that a very small percentage of claims compensated involved occupational diseases. (For example, in France there were 11.1 million accident claims recorded in 1973 and only 4,580 occupational diseases recorded.)[13]

In Belgium, where a separate compensation program handles only occupational disease claims (as will be discussed below), the bulk of claims were related to dust diseases (likely coal workers' pneumoconiosis). As shown in Appendix 2, skin conditions constituted a relatively small number, however, Barth pointed out that some cases are included under illness due to "chemical agents" and cases of chemical burns are considered "accidents" not "diseases".[14]

Table IB-2

Ontario WCB - Occupational Disease Awards 1980

Disease Category		Allowed Claims (compensation and medical aid)	Percent of Total Allowed Disease Claims
Total		**7,611**	**100.0%**
Burns		2,278	29.9%
chemical burns to eyes	654		
chemical burns to skin	548		
UVL (flash burns) to eyes	1,076		
Fume Toxicity		1,850	24.3%
smoke	413		
solvents	201		
plastic processes	186		
chlorine	153		
others	897		
Occupational Deafness		1,432	18.8%
Dermatitis		782	10.2%
oils & greases	114		
detergents & germicides	106		
plastics	86		
solvents	70		
others	406		
Inflammation of joints, tendons, muscles, arteries, etc.		579	7.6%
tendonitis, tenosynovitis, bursitis, epicondylitis	330		
carpal tunnel, ganglion	140		
VIWF	109		
Misc. diseases (including chemical conjunctivitis, allergic reactions, effects of temperature and pressure, etc.)		338	4.4%
Communicable Diseases		143	1.8%
scabies	40		
dysentery	29		
hepatitis	17		
chickenpox/herpes zoster	16		
tuberculosis	5		
other	36		
Respiratory Diseases		80	1.1%
silicosis	45		
asthmatic bronchitis	20		
asbestosis	10		
other lung conditions	5		
Cardiac Disease		65	0.8%
Malignancies. (See Chapter II for breakdown).		64	0.8%

Table IB-3

Occupational Disease Awards - 1980
British Columbia and Ontario

Disease Category	Allowed Claims—B.C.	% of Allowed Disease Claims—B.C.	Comparable Ontario %
Total	**4,212**	**100.0%**	-
Burns		26.3%	29.9%
chemical burns	615		
radiation effects	495		
Fume Toxicity		8.3%	24.3%
systemic poisoning	162		
respiratory irritation	191		
Hearing Loss	364	8.6%	18.8%
Dermatitis	376	8.9%	10.2%
Inflammation of joints, tendons, etc.	-	41.2%	7.6%
bursitis	591		
tenosynovitis	1,042		
carpal tunnel	103		
others	2		
Misc. (conjunctivitis	52	3.8%	4.4%
temperature extremes	59		
etc.).	51		
Communicable Diseases	62	2.9%	1.8%
Respiratory Diseases		0.7%	1.1%
asbestosis	11		
silicosis	17		
Cardiac Disease	17	0.4%	0.8%
Malignancies	2	0.04%	0.8%

In Switzerland, 48.5% of all occupational disease claims were for skin diseases. However, if only permanent disability industrial disease claims are considered, 75% were due to silicosis. Of all permanent disability claims, 3.1% were accounted for by occupational diseases[15] (see Appendix 2).

In Germany, home of the world's first workers' compensation system, a work injury is reported if it involves at least three lost working days, although compensation is not paid until sick insurance benefits cease after 18 days. While only 3.5% of reported injuries ultimately resulted in compensation, 16.8% of reported occupational diseases were compensated, thereby suggesting that occupational diseases reported are relatively more serious than work injuries. Overall, 8.9% of initially compensated claims in 1975 were for occupational disease. Hearing loss was the largest single source of disease; skin diseases constituted a smaller percentage as only serious and chronic dermatitis is considered an occupational disease in Germany. Of the 6,072 cases compensated, 1,208 were for silicosis, constituting 20% of the disease claims; 14 fatal cases of lung cancer were compensated, as were 7 deaths attributed to bronchial asthma. Heart cases are not considered "diseases".[16]

Thus, although methods of recording statistics are not standardized and definitions of "disease" vary considerably throughout Canada, the U.S. and Europe, in all cases the number of disease cases compensated represents only a small fraction of total cases.

Cancer in Various Canadian Jurisdictions

It is clear that serious diseases such as cancer are significantly underrepresented in workers' compensation systems throughout the various jurisdictions. In fact, Ontario's record in this regard seems to be one of the most extensive in the world. Whereas over 500 cancer claims were compensated in Ontario by the end of 1980, 137 cancer claims had been compensated in Quebec (128 lung cancers or mesothelioma due to asbestos exposure, as well as 1 lung cancer and 8 angiosarcoma of the liver attributed to vinyl chloride); 46 cancer cases had been processed in Newfoundland (for various cancers attributed to the fluorspar mines); 28 cancer claims had been allowed in Saskatchewan (including 20 lung cancers from the uranium mines and 4 mesothelioma from asbestos exposure); only 18 cancers had been allowed in B.C. (16 of which were asbestos-related lung cancer or mesothelioma) and 4 cancers (also asbestos-related) had been allowed in both Alberta and Nova Scotia.[17] The remaining Canadian jurisdictions either had no statistics available or had never compensated a cancer claim.

These figures are tabulated in column 2 of Table IB-4. The numbers are not directly comparable as the number of years in which records were kept varied considerably from one jurisdiction to another, and annual numbers of cancer claims allowed were not available in most jurisdictions. The fact that a greater share of Ontario's work force is subject to exposures in the mining industry should also be taken into account. This sector (nickel and uranium industries particularly), has accounted for a large proportion of Ontario's cancer claims (as will be shown in the following chapter).[18] It is also true that Ontario has more cancer deaths than any other province,[18] as shown in column 3 of Table IB-4.

Table IB-4

Cancer Across Canada

Canadian Jurisdiction	Total Number of Cancer Claims Compensated by 1980[a]	Number of Cancer Deaths, 1978[b]
Ontario	547	13,584
Quebec	137	10,086
Newfoundland	46 [c]	696
Saskatchewan	26	1,531
British Columbia	18	4,244
Alberta	4	2,444
Nova Scotia	4	1,543
Manitoba	n/a	1,779
New Brunswick	0	1,063
Prince Edward Island	0	198
Northwest Territories	n/a	24
Yukon	n/a	11

[a] Source: Survey conducted by the Canadian Centre for Occupational Health and Safety in answer to an enquiry. These numbers should be interpreted with caution for reasons explained in the text.

[b] Source: Statistics Canada--Cancer in Canada, 1978, Catalogue 82-207.

[c] This figure may include some rejected claims and therefore may be disproportionately high.

Barth has noted that the number of compensated respiratory cancer claims in Ontario probably exceeds the experience of any state in the U.S. He also pointed out that very few cancer claims were compensated in the various European jurisdictions surveyed.[19] Thus, whatever the reason, Ontario's record of compensating occupational cancer seems impressive in comparison to other jurisdictions.

Nonetheless, it is likely that the number of occupational disease claims compensated even in Ontario significantly understates the problem of occupational diseases. Discher et al. found that only 3% of the occupational disease cases encountered in their survey involved a workers' compensation claim,[20] and as will be shown in the following two chapters, there is reason to believe that the compensation of occupational cancer and respiratory disease in Ontario falls considerably short of the estimated prevalence of these conditions.

Some of the reasons for the undercompensation of occupational disease were presented in Part A of this chapter in relation to difficulties in assessing the dimensions of the occupational disease problem. Factors included: 1) the difficulties in determining etiology, especially in diseases which can also occur from non-workplace-related factors (the so-called "ordinary diseases" of life); 2) contention regarding the definition of "disease"; 3) lack of adequate training of medical personnel regarding occupational disease, the poor state of exposure records and the organization of occupational medicine services; and 4) the lack of scientific research on possible occupational health hazards.

A further reason for the undercompensation of occupational disease, lies in the very nature of the adjudicative framework for these claims. This will be discussed in the next section.

2. The Decision-Making Framework in Various Jurisdictions

2.1 Approaches to Determining the Work-Relatedness of Disease

Considering the broad scope of occupational disease, the etiological complexities, the diagnostic difficulties and the multitude of uncertainties alluded to above, it is not surprising that no one system has been universally acclaimed as the best approach to handling occupational disease claims. It is also not surprising that workers' compensation is the subject of considerable controversy.

Basically, there have been two broad methods of determining the work-relatedness of disease claims in the compensation systems of various jurisdictions. First, claims can be dealt with essentially as a special kind of occupational injury, each case assessed on its own merit often according to a specified definition of occupational disease. Secondly, claims can be compensated based on the use of a schedule of diseases which eases the burden on the claimant of proving occupational causation for those diseases or exposures enumerated.

A variation of this theme is the use of guidelines which set out the precise circumstances under which the claim would be allowed and therefore are generally more restrictive.

In some jurisdictions both broad methods are in use. Generally, the greater the role played by the schedule, the more union-management disputes focus on the development and amendment of the schedule rather than on the individual cases themselves.

2.2 Ontario

In Ontario, a disease may be compensated as one of the 15 "diseases" and associated "processes" listed in Schedule 3 (included as Appendix 3):

> If an employee at or immediately before the date of disablement was employed in any process mentioned in the second column of Schedule 3 and the disease contracted is the disease in the first column of the Schedule set opposite to the description of the process, the disease shall be deemed to have been due to the nature of that employment unless the contrary is proved...(S.122(9)).

If a disease is not listed in the Schedule, then the worker must show that the disease arose out of and in the course of employment, just as he or she would for an accident. However, according to the Act, a compensable disease is defined as a "disease peculiar to or characteristic of a particular industrial process, trade or occupation" (S.1(1)(n)).

Several presentations to the Weiler Inquiry have pointed out that the additional requirement that the disease be characteristic of a particular trade is a restrictive and unnecessary hurdle. Actually, the Board has exercised flexibility in this regard and has in fact compensated "ordinary" diseases not listed in the Schedule. For example, the only infectious disease listed in the Schedule is tuberculosis, yet, as shown in Table IB-2, 143 cases of infectious disease were compensated in 1980 including only 5 cases of tuberculosis. Certainly the 40 cases of scabies, 29 cases of dysentery, 17 cases of hepatitis, etc., are not "peculiar to or characteristic of" the particular employment. The WCB has even recognized several cases of male infertility as work-related (resulting from exposure to dibromochloropropane--DBCP) as well as a few cases of psychological disorders attributable to occupational stress (e.g. 2 prison guards tried and acquitted for alleged brutality). Thus, it seems that although the definition itself is restrictive, the Board uses considerable discretion in its interpretation and has indeed compensated a wide variety of diseases. (In any case, the revisions proposed for the new Act have considerably broadened the definition of industrial disease.)

2.3 *Canada*

The question of work-relatedness of disease is handled differently in each Canadian jurisdiction. Schedules of diseases were included under the Workmen's Compensation Acts of all provinces when first enacted, except New Brunswick where the Board was originally empowered to determine by regulation all the diseases to be compensated. In all the Acts, except the Acts of Manitoba and Saskatchewan and the ordinance of the Northwest Territories, a schedule of diseases has been retained.[21]

In Ontario, only 6 diseases were named in the original act. Since that time, additions have been made to bring the total to 15 classes, however, as mentioned, there have been no additions to the Schedule for some years. In contrast, in British Columbia, 75% of the disease claims processed are referred to in the schedule which was last updated January 18, 1980.[22] In Quebec, it is estimated that more than 80% of the cases are covered by the schedule which was last updated October 14, 1981.[23] Alberta's new schedule became effective January 1, 1982. As their previous schedule was not extensive or inclusive, they estimate that up to 80% of adjudication was previously based on experience and various policies rather than schedules.[24]

In Alberta, Saskatchewan, Manitoba, New Brunswick, Newfoundland as well as in Ontario, wider coverage of industrial disease is also provided by a broadened definition of "accident". In British Columbia, New Brunswick, Quebec, as well as Ontario, the Board may also recognize any disease as being a disease "peculiar to" or "characteristic of" a particular industrial process, trade or occupation.

It is difficult to determine how rigidly these definitions are adhered to, as illustrated for Ontario above. Suffice it to say that while some jurisdictions have shown a clear preference to adjudicate each case on its own merits, other jurisdictions prefer to use a schedule.

2.4 *The U.S.*

In some U.S. jurisdictions schedules have been expanded. However, rather than expanding, the recent trend is toward general coverage either by adding a "catch-all" phrase to cover the diseases not listed, or by abandoning the schedule altogether and replacing it with a general definition. (Only 9 U.S. states still employ a schedule.) However, as concluded by the U.S. Policy, Evaluation and Research Committee (Labour) 1979:

> The general definitions are usually so hedged with restrictive phrases and conditions that they do not serve the purpose of actually providing coverage of all work-related diseases .... one would suppose that the purpose of defining 'occupational disease' would be to delineate which diseases are sufficiently and clearly work-related as to entitle the victims to compensation. A close review of the current statutes can only lead one to believe that the real purpose is to limit the number of claims, especially of the chronic, long-term (and probably costly) variety, which are admitted into the system.[25]

Twenty-one states use the restriction "peculiar to" (like Ontario). Similarly, about 30 states exclude "ordinary disease of life". The U.S. recommendation however, specifically outlined the need to "leave the 'peculiar' and 'increased' risk tests behind and adopt an 'actual' risk test based on the simple fact that the disease was indeed due to or connected in a significant way with the employment."[26]

The purpose of schedules is to give the worker the benefit of a statutory presumption in favour of allowing his or her claim. The difficulty of proving causal relationship to the employment is well-known, particularly with respect to diseases of non-specific origin. Rebuttable presumptions are by no means uncommon in workers' compensation laws. They may be very general or very specific, broad or limited. A number of U.S. states including New York, Massachusetts and Florida contain a "general" presumption that, in the absence of substantial evidence to the contrary, the claim comes within the provision of the Act. However, Florida, like certain Canadian provinces including Ontario, specifically states that this does not apply to a claim for occupational disease.

Another type of presumption found in several states including Illinois, Indiana and Missouri provides help in establishing proof of exposure: "An employee shall be conclusively deemed to have been exposed to the hazards of an occupational disease when for any length of time, however short, he or she is employed in an occupation or process in which the hazard of the disease exists."[27]

A number of U.S. states (Alabama, Illinois, Kentucky, Montana, North Dakota, Tennessee, Utah and Virginia) have adopted presumptions for coal miners suffering from black lung similar to the Federal Black Lung Act, i.e. the claimant is presumed to be suffering from black lung (and that the disease is work-related) if he meets certain conditions of disability and work history in coal mines. (If he worked 15 or more years in coal mines, a claimant need only present evidence that he is totally disabled by a respiratory impairment...even if the chest X-ray is negative, etc.). The House of Representatives actually passed a further reform establishing an irrebuttable presumption of total disability based solely on years of coal mine employment, however the Senate failed to pass it.

Another group of states have provided presumptions for fire-fighters and/or law enforcement officers who have suffered disability or death from heart or lung diseases. Many states invoke the presumption only if employment exceeds a given number of years. It should be noted that Manitoba invoked a presumption for fire-fighters who suffer heart or lung disease in the course of their work; British Columbia and New Brunswick also have special presumptions for fire-fighters in their Acts.

In Newfoundland, a statutory presumption exists for fluorspar miners who contract certain respiratory ailments:

> Individuals who at any time worked underground in the fluorspar mines...and who are disabled because of lung cancer or silicosis should receive compensation...That individuals who worked in the mines prior to 1960 who developed disability from a chronic obstructive pulmonary disease of any kind whatsoever which renders them physically unfit to work again in a fluorspar mine...should be compensated for total disability....[28]

2.5 *Europe*

In his recent study, Professor Peter Barth examined workers' compensation systems in various European countries. The British system relies heavily on a schedule of diseases which as of January 1976 contained a listing of 49 sets of diseases, (and now includes 52). The only way in which an unscheduled disease could be compensated was if it was termed an injury due to accident. If an unscheduled disease was caused by prolonged exposure to a hazard, it would not be compensable. Thus, vibration-induced white finger, for example, is not included in the schedule and is therefore not compensable. There are no minimum or recent exposure rules that must be met except in a few circumstances such as occupational deafness, which requires a minimum of 20 years' occupational noise exposure and must be filed within a year of leaving employment, and byssinosis, which requires 10 years' exposure. The responsibility for modifying the schedule lies with the multipartite Industrial Injuries Advisory Council which consists of 4 union appointees, 4 management representatives and 8 drawn from other areas, mainly the medical community. Barth points out that while the British Trade Union Congress continues to work to widen the schedule, recently pushing to include bronchitis, for example, they accept the reality that a complete listing of all possible diseases and employments would undermine the very strong presumption afforded by the schedule, with which they are quite satisfied.[29]

Barth found that France also uses a highly detailed and comprehensive schedule, which at the time contained 64 tables, each covering a different class of substances or diseases. Each table lists specific illnesses, time period from last exposure in which the workers' compensation system is liable, and a list of tasks which would be presumed to be linked to the disease. Minimum exposure rules do exist for a few listed diseases, but can occasionally be waived. Revisions in the schedule occur frequently and are the responsibility of an Industrial Hygiene Commission comprised of civil servants, technical specialists and representatives of labour and management. The Commission has a subcommittee on industrial diseases which in turn has working groups composed of physicians and other health personnel. (Barth notes that when several factors, including pre-existing conditions, contribute to causing a disease, as long as one of the factors is drawn from the Schedule, the worker will be compensated. He also points out, though, that the presence of the other factors can reduce the extent of the disability award.[30] (The manner in which this issue is addressed in Ontario will be discussed at length in Chapter III.)

Occupational diseases in Belgium are compensated in a system totally distinct from that which compensates occupational injuries. Again, a schedule of diseases is exclusively used. Apparently similar to that of Switzerland, the first portion of its schedule lists substances or classes of substances and for most, identifies industries (or occupations) in which diseases would be covered. For these substances, specific diseases are not listed. The second part of the list covers skin illnesses caused by substances not listed elsewhere; the third part covers illnesses caused by inhalation of substances and agents not listed elsewhere; the next section covers infectious and parasitic diseases with specific industries and occupations identified; the final section covers illnesses caused by physical agents. The Swiss schedule is almost identical to the International Labour Organization (ILO) schedule, and, in contrast to the detailed and explicit schedule in countries such as France, is considered to be a model of simplicity. As an open-ended provision exists, 27.2% of all compensated occupational disease cases come from illnesses or hazards that are not in the schedule but are adjudicated on their own merit.[31]

Denmark also has a "mixed system" in virtual conformity with the recommendations of the European Economic Community (EEC). A worker's disease is considered occupational when, according to medical and technical opinion, he or she had a higher risk of contracting it than an ordinary person. A schedule exists linking certain diseases to specific occupations or industries, but the latter are considered samples and are not exclusive.[32]

Germany's schedule is considered strict by European standards. Although there is an open-ended provision for claims, few have been compensated that were not in the schedule.[33]

In the Netherlands, the workers' compensation system was abolished in 1967 and replaced by a comprehensive insurance plan, protecting workers against disability regardless of the job-connection.[34]

In Sweden, the term "work-injuries" includes all injuries and diseases caused by accidents or other harmful influences at work. As examples of "other harmful influences", the drafting documents of the Act refer to substances, energy radiation, vibration, noise, low or high temperatures, damp and heavy drafts, as well as monotonous, unusual or unusually strenuous movements or work postures and repeated or unusual pressure. Mentally strenuous conditions directly associated with work may also come under the scope of the Act. Special rules apply, however, to infectious diseases, which only qualify under the workers' compensation scheme if they are included in a special schedule.

Workers' compensation is co-ordinated with general health insurance for the first 90 days of disability. This means that during this period the injured worker receives the same health insurance benefits as others, corresponding to 90% of his or her earned income. No consideration is given to the possible work-relatedness of the claim during this period. After that period, the Swedes still note that "legal practice demands quite a high degree of probability or at least plausible grounds in order, for instance, for a substance or some other factor at work to be rated per se as a potential cause of injury."

The workers' compensation system, like general social insurance, employs an economic concept of invalidity. This means that the system does not provide benefits for incapacity or pain and suffering without economic loss. Workers, however, are able to obtain compensation for this kind of so-called non-financial damage under special social security insurance agreements which have been concluded between associations of employers and trade unions and which apparently include practically all workers in Sweden.[35]

2.6 *Comparative Assessment*

Thus various permutations and combinations of approaches to compensating victims of industrial disease have been applied across North America and in Europe. As record keeping varies substantially from one jurisdiction to the next, it is difficult to compare the overall results using the various systems. In Quebec and Manitoba, as well as here in Ontario, the data recorded do not even allow a comparison of the total number of industrial disease claims allowed with the number rejected. In Alberta, 1,181 industrial disease claims were received in 1980; of these 916 or 77.6% were allowed. British Columbia indicates that 4,152 of the 4,289 claims received in 1980, or 96.4%, were allowed. Of the 238 industrial disease claims reported in Saskatchewan in 1980, 218 or 91.5% were allowed.[36] Thus, no system seems obviously superior, however, the data bases and the possible differences in definition of industrial disease make any conclusions very tenuous. (For example, as was noted in Section 1, Alberta does not seem to include chemical burns as a "disease". Since this category would tend to have a relatively high allowance rate, its exclusion would tend to artifically lower the allowance rate there in comparison to jurisdictions which include this category.)

In Europe, as well, the data are difficult to assess comparatively. In Belgium, where a separate system handles occupational disease claims through the exclusive use of a schedule, it was indicated that 6,557 of the 15,167 claims received in 1974 (43.2%) were rejected. Oddly, Barth notes that only 2% of the industrial disease claims were rejected in Switzerland, where a similar, but not exclusive schedule is used.[37]

The U.S. Department of Labour investigating the workers' compensation system in the U.S., attempted to analyze the number of disabling occupational disease cases compensated in various industrialized countries, weighted to reflect the size of the U.S. work force. As shown in Table IB-5, Ontario ranked second, ahead of Belgium, Switzerland, Britain, France and the U.S. In fact, Ontario compensated four times the relative number of industrial disease claims compensated in the U.S. However, Ontario compensated only slightly more than half the number of occupational disease cases compensated in Sweden.[38] Given the

Table IB-5

Workers' Compensation for Disabling Occupational Diseases[a]

Jurisdiction	No. of Disabling O.D. Cases Compensated[b]	No. Compensated Claims Weighted to Reflect Size of U.S. Work Force
Sweden	16,000	340,000
Ontario	5,032	140,000
Belgium	5,737	140,000
Switzerland	4,047	125,000
Great Britain	14,499	50,000
France	8,080	40,000
U.S.A.	30,000	30,000

[a] Reprinted from U.S. DOL Interim Report to Congress on Occupational Diseases, 1980, p. 72. Analysis and tabulations prepared by ASPER, U.S. DOL.

[b] According to the Interim Report, the numbers of occupational disease claims paid were taken from several sources and reference years. Those for Belgium (1974), France (1973) and Great Britain (1975) were from Barth, P. and Hunt, A. Workers' Compensation and Work-Related Illnesses and Diseases. Data for Ontario and Sweden (1977) came from unpublished tables supplied by their respective Workers' Compensation Boards.

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exclusion of short-term and non-wage-loss claims from these statistics, this seems quite remarkable, if in fact this methodology has any validity.

Even if it were possible to clearly indicate which system seems to work best, it must be realized that the attitudes and institutions that exist in one jurisdiction, as well as the complementary programs that operate jointly within it, may well be the essential factors in allowing the success. Nonetheless, it seems that the existence of a schedule has definite advantages, especially where accompanied by open-ended provisions for adjudication of cases which do not meet the specifications of the schedule.

Ontario indeed has a "mixed" system of this sort. However, numerous problems have been identified which warrant attention. Some of these will be briefly illustrated in the following section and expanded upon in the following chapters.

3. The Problems Introduced

As demonstrated above, the Workers' Compensation Act of Ontario allows the Board to exercise considerable flexibility and to use its discretion with respect to waiving the "peculiar to and characteristic of" requirement. Nonetheless, often the Board has chosen not to grant compensation for common diseases of life to which occupational factors may have contributed; and often the Board has rigidly adhered to its own guidelines even where common sense seemed to indicate that flexibility would have been in order.

In some cases, the onus on the claimant to provide proof of exposure and proof of the work-relatedness of his or her disability is such that the claimant almost has to be an industrial hygienist, an epidemiologist, a detective and a lawyer in order to fight his or her claim.

The following cases begin to illustrate some of the problems that plague the Workers' Compensation Board. The points raised here will be elaborated upon in subsequent chapters.

3.1 Degenerative Disc Disease and Heavy Labour

As explained in Appendix 4, degenerative disc disease (DDD) is not characteristic of any particular trade or occupation, but rather results from the wear and tear of life. It is often associated with the aging process and hereditary factors have been implicated. However, it has also been shown that stresses such as heavy lifting, bending and various postures and movements demanded in a given job can clearly contribute to the development of the disease. Epidemiological studies have also revealed that various features of DDD are more prevalent in certain occupations, including construction, and have shown, for example, that low back pain is a major cause of retirement in this occupation. (See Appendix 4)

Despite this evidence, or perhaps because of its complexity, degenerative disc disease is not considered an occupational disease in Ontario. In fact, the finding of DDD in a worker subject to strenuous work can even negatively influence the outcome of the claim. The following case is typical.

Claim No. 1: In March 1974, Mr. G., age 30, a labourer employed for about 4 1/2 months with K. Construction Ltd., strained his lower back while lifting the end of a piece of structural steel. Treatment was conservative and he returned two weeks later to resume heavy work. Since this incident, Mr. G. suffered recurrence of back pain on an average of once or twice per year lasting three or four days each time. He apparently complained to co-workers and foremen regarding his back in all the companies for which he had worked throughout this period. In September of 1980 he noted gradually increasing backache while performing his job, digging underground so that pipes could be placed. The foreman advised him to work at a slow pace and to rest; however, he continued to work the full day despite the increasing pain. Finally, he sought medical attention and an acute lumbar disc protrusion was diagnosed. He was treated with physiotherapy and a corset, and remained off work until January 1981. His family doctor referred to this backache as a "recurrence of the 1974 injury" and compensation was sought.

There was no dispute that Mr. G. had a significant back problem, nor was it questioned that his job was extremely heavy. His foreman's statement reflected that Mr. G.'s job "involved a lot of shovelling and that he was working in a bent-over position almost all of the time." It was also reported that "it was obvious from the way he walked and worked that he was in a lot of discomfort."

The specialist consulted by the WCB regarding this case noted that X-rays of the lumbar spine showed gross abnormalities consistent with DDD. He stated that:

> Although physical examination indicated minor nerve root irritation, this is not unusual with an exacerbation of DDD...One could assume that this patient had a serious single disc injury over this length of time and with the amount of heavy work he was doing that he would have developed more specific areas of neurological change. His tenderness, however, appeared to be from the second to the fifth lumbar vertebra and this is in keeping with DDD.... In my opinion, since there have been no further specific incidents or injuries to indicate a new lumbar incident, I would conclude that the September 1980 layoff and the incidents previous to this were not related specifically to the incident of May 1974.

The claim was denied, as was the appeal.

Commentary: The only issue addressed in this case was whether or not one specific injury was causally related to the recurrent back problem. The possibility that the DDD itself was work-related was never even considered. The worker understandably assumed that because his back problem began at the time of the 1974 injury and his discomfort was similar in nature since that time, that his back problem was related to the accident. As the Board consultant concluded that this was not so, the claim was denied. The practice of adjudicating only on the question of eligibility for compensation with respect to the particular disease (or, in this case, "injury") specified by the claimant was observed in many of the cases reviewed for this Inquiry. This practice is certainly undesirable and inconsistent with the intent of workers' compensation.

Secondly, description of the claimant's work history was extremely scant. In fact, the only real job description was that provided in the foreman's statement. The disproportionate attention given to medical detail as compared to details of the work situation is also characteristic of many of the claims reviewed, as is a third issue introduced by this case, that of conflicting medical opinions.

The narrow approach in adjudicating this claim, the limited analysis of workplace factors, and the role of medical professionals are procedural issues which can be rectified with appropriate measures. The more difficult conceptual problem is how common diseases such as DDD which are not strictly "occupational diseases" but in which occupational factors can play some role, can be dealt with by the compensation system. Decision No. 99 rendered by a British Columbia Appeal Board in March 1975, addressed this issue:

> Where the degeneration is of a kind that affects the population at large, it would be impossible for the Board to attempt a measurement of the significance of each occupation on each kind of degeneration to determine whether a particular occupation had any effect in advancing the pace of degeneration compared with other occupations, or compared with a life of leisure....For a disability to be compensable, it must appear that the work activity brought about a disability that would probably not otherwise have occurred, or that the work activity significantly advanced the development of a disability that would otherwise probably not have occurred until later....

It was concluded that DDD did not meet this latter criterion and this claim too was denied. Although it seems that enough scientific evidence may soon exist to warrant a change in this policy with respect to DDD, the dilemma raised is a recurring theme throughout this report and is one of the major issues in need of resolution.

3.2 Chronic Poisoning from Chemical Exposure

The discretionary power of the Board to overrule the "peculiar to and characteristic of" as well as the "immediately prior to" requirement is paralleled by its flexibility in interpreting the schedule of diseases. Even when a person has a listed "disease" and was exposed to the corresponding "process", compensation is not necessarily granted by the Board. The presumption of work-relatedness can be refuted if it can be shown that the disease was not due to the nature of employment.

Moreover, it is not always easy for the claimant to prove exposure to one of the enumerated processes, nor is it a simple matter to determine if a disease indeed fits the description of the "disease" prescribed. The following case illustrates the difficulties in ascertaining accurate exposure data as well as the problems in proving that the disease was indeed caused by this exposure.

Claim 2: This claim was filed for Mr. M., attributing his development of chronic hepatitis to exposure to toxic chemicals at work. The claimant had been employed at a small tool and mould company for seven years and had given a history of intermittent nosebleeds and progressive fatigue for about a year and a half prior to seeking medical attention. In November 1979 his chronic liver disease was diagnosed and his doctor sent a letter to his employer stating: "It is felt that further exposure may cause progression of his liver disease. It is advisable that he be placed in a job situation where exposure is minimal." Compensation was sought. One senior Board medical consultant urged a complete investigation of all chemicals to which he was exposed, but noted that "it was not thought likely that the claim will be found to have merit."

At work he had been exposed to a variety of chemicals. Reference was made in the file to possible exposure to carbon tetrachloride, trichloroethane, vinyl chloride and styrene, all known liver toxins. It was noted that the Occupational Health Branch's report indicated the presence of the latter two compounds, but no indication was given as to the degree of exposure; no evidence was ever provided as to exposure to the former two components, or, in fact, to confirm the latter substances.

Well-documented daily exposure to kerosene was, however, confirmed but there was much confusion as to the extent of exposure and to its exact composition, specifically, whether or not it contained benzene. There was even confusion as to the origin of this solvent, as reflected by the testimony of one industrial hygienist: "The general manager thought it was a product of company A. The person ordering materials suggested it came from company B and company B apparently gets its petroleum solvents from a company C. And company C, I'm pretty sure, thought it might have been a product of a fourth company as far as they were concerned." Exposure was also confirmed to ethyl and methyl alcohol, capable of potentiating other hepatotoxic effects, and butyl cellusolve, an ether compound known from animal experiments to have a toxic effect on the liver.

In April 1980, the claim was rejected for the following reasons:

1. the claimant worked 8 years with no problems;
2. there was minimal exposure to chemicals;
3. the area was well-ventilated;
4. no other workers had similar conditions;
5. the injured employee could have reduced his exposure to the chemicals by using water as a remover rather than kerosene; and
6. his previous medical history indicated that he had been treated for a congenital infectious disease as a child.

An appeal ensued. Following the hearing before an appeals adjudicator, further investigations regarding the chemical exposure were conducted by both the WCB and the Occupational Health Branch of the Ministry of Labour, and the components of the various trade name products were ascertained. In June 1981, the appeal adjudicator concluded that "the evidence has not established that Mr. M. was exposed to hepatotoxic chemicals in the course of his employment and therefore there is no basis for considering his chronic hepatitis to have been related to his employment."

A further appeal was launched. More evidence regarding the hepatoxicity of the exposure chemicals was gathered by the claimant's representatives and the claimant sought advice from the Occupational Health Clinic at St. Michael's Hospital. The occupational medicine specialists there concluded that the clinical pattern of the disease, as well as the evidence against it being either of viral etiology or due to alcohol-abuse (the other known causes of this disease) suggested the likelihood of occupational origin. One doctor noted:

> The most likely explanation, therefore, is a prolonged exposure to not severely-hepatotoxic agents. Occupational exposures are immediately the most likely to fit into this category. This man did have exposure on a consistent basis to ethyl and methyl alcohol and other solvents at work. Alcohols will potentiate the hepatotoxic effect of other agents. The aromatic components of Stoddard's solvent or kerosene could be hepatotoxic. Butyl

> cellusolve in the dye he was using will, if absorbed through the skin, exert a hepatotoxic effect. His complete exposure history appears to be unknown. Although it is reported he could have used less solvent on his job, he appears in fact to have had a fair amount of exposure, including wetting of his work clothes. Therefore, this man's liver disease is most likely due to occupational exposures, some of it at least due to solvent exposure. I do not have a better medical explanation.

Subsequent to the hearing, held in February 1982, it was decided that the case would be referred to an independent specialist. As of August 1982, the case was still pending, and the claimant had spent over 2 years with no reasonable income.

Commentary: The original rationale for rejection of the case illustrates how decisions may be based on completely irrelevant factors, such as reasons number 1 and 4; the relevance of reason 6 is certainly questionable medically and otherwise; reason 5, that the claimant was partly responsible for the extent of his exposure, is not only irrelevant but contrary to the intent of workers' compensation; and consequently the conclusions leading to reasons 2 and 3, which were hotly disputed throughout the case, were not adequately explained.

Moreover, this case clearly illustrates where the onus of proof lies. It was incumbent on the claimant first to establish evidence of exposure and secondly to provide the scientific evidence that this exposure could lead to the disease in question. Despite the complete absence of any competing explanation for the disease, the proof of occupational causation was considered inadequate. Many cases raising these same issues will be presented in the following chapters.

3.3 Noise-Induced Hearing Loss

In the Survey of Industrial Disease Claims performed for this Inquiry, it was found that one-third of the claims for noise-induced hearing loss were rejected (see Tables A and B of Appendix 5). Although the rejection rate for cancer and respiratory disease was almost twice as high, the rejection of hearing loss claims is much higher than the 2% rejection rate of total claims received. As noise-induced hearing loss is one of the more easily recognizable occupational diseases, with characteristic audiogram findings, this high rejection rate is of concern. On reviewing a sample of rejected cases, it was found that the denials related largely to failure to meet one or both of the requirements set out in the guidelines:

(a) exposure to a noise level of a minimum of 90 decibels for an 8-hour period per day for at least 5 years; and

(b) minimum hearing loss in both ears of 25 dB to qualify for medical aid benefits and 35 dB to qualify for compensation.

The following case, allowed on appeal, is indicative of the restrictive effect of these guidelines:

Claim No. 3: This worker spent 40 years employed in various noisy jobs at a forge, and had been wearing hearing protection only for the last 4 of these years. The medical report revealed this to be a classic case of industrial hearing loss. He had been gradually losing his hearing in both ears, he had no history of head or ear injury, no history of significant non-occupational noise exposure, no history of exposure to ototoxic drugs, no history of any other ear disease, and no family history of ear disease. It was also noted that his audiogram was typical of noise-induced hearing loss. The original rejection was based on noise exposure records that failed to add up to the required noise level. On re-investigation following the appeal hearing, it was found that in fact the noise exposure was "sufficient".

Commentary: It was pointed out in presentations to the Weiler Inquiry that the requirement that a worker prove sufficient noise exposure is analagous to requiring a worker to prove that the floor on which he slipped was sufficiently slippery to have caused his accident. It has been argued that since the WCB clearly does not require the latter, it should not require the former. Moreover, 90 dB is not a safe level below which deafness could not result. It is well-accepted that cases of hearing loss have occurred and will continue to occur at noise exposure below this level. Therefore, reference to this level in adjudicating claims where there is clear evidence of noise-induced hearing loss with no other plausible explanation, is clearly illogical. Further, it accounts for considerable delay in the adjudication of claims. The mean time from claim set-up to initial decision was found in our survey to be 24.1 weeks (see Table C, Appendix 5).

The requirement for 'sufficient disability' is also inconsistent with the requirements for accident victims, and has resulted in undue hardships as illustrated by the following case.

Claim No. 4: The reason initially given for rejection of this claim was that although 'enough deafness' had been documented in the audiogram, there had been insufficient evidence of exposure. The claim was appealed; the claimant's representative referred to other cases from the same workplace that had been allowed and urged verification of the noise exposure estimates. On re-investigation, it was found that indeed there had been sufficient noise exposure and the appeals adjudicator consequently allowed the claim. On assessment for a permanent disability award, he was found not to have sufficient deafness for allowance. Due to the discrepancy between audiograms, further testing was arranged, which unfortunately for the claimant, substantiated that he had a bilateral hearing loss of only 20 dB. Although two specialists agreed that a hearing aid would be helpful, the Appeal Board denied the request. A complaint was consequently filed with the Ombudsman in which it was contended that it was unreasonable to deny benefits for medical aid for hearing loss clearly of industrial origin, and urged that the claim be examined on its own merits. On last review, the response was still pending.

Commentary: Since the claimant's hearing loss was attributed to his employment it seems unfair that failure to meet a Board guideline disqualified him from receiving a hearing aid. Indeed, there is no provision in the statute which prohibits allowing this claim. Thus the rejection was a matter of administrative practice rather than legal constraint.

3.4 Diseases Induced by Occupational Stress

There is growing recognition not only of the hazardous effects of noxious chemicals, biological, physical and biomechanical factors, but also the potentially deleterious health effects of psychological or sociotechnical aspects of the work environment. Mental stress has long been associated with a variety of illnesses and there are an increasing number of publications on the effect of various factors in the workplace on the psychological and physical well-being of workers. (See Appendix 6.) The etiological complexities involving work-related mental stress have already begun to plague the compensation system with regard to two main categories of disease: cardiovascular diseases and mental illnesses; other stress-related diseases, such as peptic ulcer may also pose problems in this regard.

The data recording system of the Ontario Workers' Compensation Board does not allow an appraisal of the number of claims that have been made to the Board involving mental stress. Two cases directly contending that occupational stress caused the disability in question were encountered in the process of reviewing cases for this study. One claim was from a 50-year-old physician of 23 years who had a cerebrovascular accident (stroke) while making a house call after night office hours. His contention that excess work and extremely long hours were causally-related to his disability was denied. The second case was a claim for fatal benefits from the widow of a cameraman who had a myocardial infarction (heart attack) while filming at the mayor's office. Here again it was contended that various occupational stresses, including long working hours and job pressure, were causally related to the death, but again the claim was denied. (This case is included in Section 3 of Chapter IV in a larger discussion of WCB policy regarding cardiac claims.)

The compensation of a Quebec worker whose anxiety reaction was attributed to job harassment was noted in Part A, as were several cases which were compensated here in Ontario. A case recently before the WCB of British Columbia received considerable attention. The claimant contended that his alcoholism and consequent cirrhosis of the liver resulted from the working conditions which he was forced to endure - specifically, prolonged periods of isolation in logging camps. The potential implications of recognizing alcoholism as an occupational disease are far-reaching.

Employer responsibility for alcoholism is a topic of a major work soon to be published by the Addiction Research Foundation.[39] It contends that many loss-causing events, including those associated with cardiovascular disease and alcohol abuse, are to some extent tied up with the very way in which work is organized. While it is noted that the Ontario WCB is very concerned about alcoholism as a contributory factor in many compensable accidents, the authors point out that the seminars conducted by the WCB deal essentially with the issues that arise in dealing with alcoholism upon an individual basis once the symptoms are visible, but fail to address the cause of the condition. They suggest that if the compensation system would acknowledge alcoholism as compensable under certain conditions, it would encourage a shift of emphasis toward preventive measures, specifically with respect to factors over which employers have some degree of control (see Appendix 6).

While some people turn to alcohol in times of stress, work-related or otherwise, other people develop other sorts of stress-related diseases. It has been noted that mental strain triggered by work stress is, in fact, now compensable in several jurisdictions. One well-publicized case involved the compensation awarded in Michigan to a machine operator on an assembly line who developed a psychosis as a result of the emotional pressure he encountered in his daily work. Medical reports indicated that the claimant "had a personality predisposition towards development of this illness for a number of years" and the anxiety he suffered because of his job pressure was "the straw that broke the camel's back". The court observed that the emotional pressures produced by his production-line employment did not differ in any respect from the emotional pressures encountered by his fellow workers in similar employment, but decided that the evidence had indeed established a causal connection between the series of mental stimuli and his disability, and therefore compensation was awarded.[40] Similarly, a California court awarded compensation to a fire-fighter who developed a psychoneurosis as a result of the cumulative effect of each day's stresses and strains.[41]

In common with alcoholism and other stress-related diseases, cardiovascular disease presents the problems of being an ordinary multicausal disease of life. Factors such as smoking, excessive drinking, dietary habits and lack of exercise as well as high levels of stress have been associated with an increased risk of cardiovascular disease. It has been pointed out that compensation law surrounding cardiovascular disease is characterized by a concern to protect the system from unworthy claims. As diseases of the heart account for over 22,000 annual deaths in Ontario alone, with cerebrovascular diseases accounting for another 5,600 (see Appendix 1), this concern is not surprising.

Some jurisdictions do, however, allow these sorts of claims. A case was cited by Barth of compensation awarded to the survivor of a maintenance worker whose "anxieties, fears and insecurities, frustration and guilt feelings" in relation to his job were deemed causally-related to his death. He had been responsible for preventing corrosion of airplanes and his employer had threatened to fire him if he had not repaired a particular plane by a certain deadline.[42] Other cases of compensation for heart attacks attributable to occupational stress included the compensation of a worker whose attack was precipitated by an argument he had with his supervisor,[43] and the compensation of an attorney whose heart attack was deemed to be precipitated by a seven-week period of strenuous litigation.[44]

As will be discussed in Chapter IV, the medical staff of the Ontario WCB reviewed the literature on the subject of stress and cardiovascular disease and concluded that scientific justification for compensation of these cases did not exist. This, in fact, is consistent with the findings of another study in which, using the medical criteria for compensability established by the American Heart Association, it was found that half of the successful heart claims in Wisconsin could not be justified medically.[45]

In another study, conducted by the California Heart Association, five cardiologists examined the files of 319 cases. In only 47 of these cases did the entire panel agree about causality. In 215 cases, only three doctors could agree and in 48 cases, fewer than three agreed. More disturbing was the finding that when 101 records were reviewed by the panel, without their knowledge that these were the same files previously examined, in 30% of the cases they reversed their original decisions.[46]

This finding led Barth to conclude that "...to ask anyone, either a compensation administrator or a physician, to evaluate the cause of a disease whose etiology is almost always unknown for the purpose of satisfying a legal proceeding, results in arbitrary, capricious and inconsistent determinations that are ultimately based on secondary or tertiary considerations."[47]

Thus, the dilemma surrounding the compensation of multifactorial, stress-related diseases provides yet another indication of the difficulties which arise in the medical assessment of causality and therefore compensability of diseases of controversial etiology. While research into the relative role of each of the causative factors in these diseases must continue, it must be recognized that science presently offers limited assistance in the search for appropriate criteria for compensation.

With the problems of compensating occupational diseases introduced, the following two chapters will extensively review the issues pertinent to the compensation of occupational cancer and respiratory diseases respectively.

4. The Issues Summarized

1. The serious chronic diseases which account for the bulk of morbidity and mortality in the Ontario population account for only a tiny percentage of the workers' compensation claims filed annually, and an even more minute fraction of the claims allowed.

   The experience for Ontario is similar to that in other jurisdictions across Canada, the U.S. and Europe. Lack of standardized definitions and recording methods precludes accurate direct comparisons, however, Ontario's record of compensating serious diseases such as occupational cancer seems impressive in comparison to other jurisdictions.

2. Various approaches exist to determining the work-relatedness of disease claims. However, basically claims can either be adjudicated on their own merit, or eligibility for compensation can be guided by a schedule of diseases. Policy guidelines are a variation of this second option but usually are more detailed and hence restrictive, and lack the legal formality of schedules.

   Various combinations of these approaches exist in the different jurisdictions across Canada, the U.S. and Europe. No system stands out as being obviously superior. The "mixed system", wherein schedules (or guidelines) are used in conjunction with open-ended provisions for adjudication of cases which do not meet the specification of the schedule, has definite advantages. Ontario has such a system.

3. On reviewing claims relating to diseases as diverse as degenerative disc disease, chronic hepatitis, noise-induced hearing loss, and diseases of psychological stress, several problem areas in the policy and procedures of the Ontario Workers' Compensation Board have been introduced. These include: 1) the practice of adjudicating solely on the basis of the disease

specifically identified by the claimant, 2) heavy emphasis on the claimant's medical history as compared to the interaction of the worker with the workplace or the details of the workplace itself, 3) confusion regarding which factors are relevant to the adjudication process, 4) the time-consuming requirement of assessing if exposure surpassed an arbitrarily set level, 5) the requirement that the claimant pass an arbitrary test of impairment in order to be eligible for medical aid, 6) the difficulty in ascertaining exposure data and then determining the likelihood of this exposure having caused a poorly studied, non-specific disease, and, most importantly, 7) the difficult conceptual problem of assessing the role of occupational factors in multifactorial diseases.

These issues will be examined in greater detail in the following chapters.

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APPENDIX 1

MORBIDITY AND MORTALITY STATISTICS - ONTARIO

Table A

International Classification of Diseases (Adapted 8th Rev.) "A" List	AGE GROUPS AND CAUSES OF DEATH	Deaths from Specified Causes			
		In Age Groups		At All Ages	
		Number	Percent	Number	Percent
	5-14 years	440			
AE 138 - AE 150	Accidental and violent deaths	257	58.4	4,994	5.1
A 45 - A 60	Cancer (all forms)	61	13.9	13,584	0.4
A 126 - A 130	Congenital anomalies	34	7.7	507	6.7
A 90 - A 93	Influenza, pneumonia, bronchitis, emphysema and asthma	10	2.3	2,834	0.4
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	8	1.8	22,149	0.03
	Other causes	70	15.9		
	15-19 years	661			
AE 138 - AE 150	Accidental and violent deaths	518	78.4	4,994	10.4
A 45 - A 60	Cancer (all forms)	49	7.4	13,584	0.4
A 126 - A 130	Congenital anomalies	12	1.8	507	2.4
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	9	1.4	22,149	0.04
A 90 - A 93	Influenza, pneumonia, bronchitis, emphysema and asthma	8	1.2	2,834	0.3
	Other causes	65	9.8		
	20-24 years	773			
AE 138 - AE 150	Accidental and violent deaths	599	77.5	4,994	12.0
A 45 - A 60	Cancer (all forms)	50	6.5	13,584	0.4
A 126 - A 130	Congenital anomalies	12	1.6	507	2.4
A 90 - A 93	Influenza, pneumonia, bronchitis, emphysema and asthma	11	1.4	2,834	0.4
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	10	1.3	22,149	0.04
	Other causes	91	11.8		
	25-34 years	1,215			
AE 138 - AE 150	Accidental and violent deaths	730	60.1	4,994	14.6
A 45 - A 60	Cancer (all forms)	196	16.1	13,584	1.4
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	65	5.3	22,149	0.3
A 85	Cerebrovascular disease	32	2.6	5,610	0.6
A 90 - A 93	Influenza, pneumonia, bronchitis, emphysema and asthma	26	2.1	2,834	0.9
	Other causes	166	13.7		
	35-44 years	1,772			
AE 138 - AE 150	Accidental and violent deaths	532	30.0	4,994	10.7
A 45 - A 60	Cancer (all forms)	416	23.5	13,584	3.1
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	375	21.2	22,149	1.7
A 102	Cirrhosis of the liver	83	4.7	1,020	8.1
A 85	Cerebrovascular disease	69	3.9	5,610	1.2
	Other causes	297	16.8		
	45-54 years	4,856			
A 45 - A 60	Cancer (all forms)	1,627	33.5	13,584	12.0
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	1,452	29.9	22,149	6.6
AE 138 - AE 150	Accidental and violent deaths	607	12.5	4,994	12.2
A 102	Cirrhosis of the liver	281	5.8	1,020	27.5
A 85	Cerebrovascular disease	200	4.1	5,610	3.6
	Other causes	689	14.2		
	55-64 years	9,246			
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	3,405	36.8	22,149	15.4
A 45 - A 60	Cancer (all forms)	2,968	32.1	13,584	21.8
AE 138 - AE 150	Accidental and violent deaths	521	5.6	4,994	10.4
A 85	Cerebrovascular disease	484	5.2	5,610	8.6
A 102	Cirrhosis of the liver	329	3.6	1,020	32.3
	Other causes	1,539	16.6		
	65-74 years	14,454			
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	5,705	39.5	22,149	25.8
A 45 - A 60	Cancer (all forms)	4,048	28.0	13,584	29.8
A 85	Cerebrovascular disease	1,174	8.1	5,610	20.9
A 90 - A 93	Influenza, pneumonia, bronchitis, emphysema and asthma	623	4.3	2,834	22.0
AE 138 - AE 150	Accidental and violent deaths	433	3.0	4,994	8.7
	Other causes	2,471	17.1		
	75 years and over	26,070			
A 80A, A 81, A 82A, A 83, A 84	Diseases of the heart	11,107	42.6	22,149	50.1
A 45 - A 60	Cancer (all forms)	4,135	15.9	13,584	30.4
A 85	Cerebrovascular disease	3,624	13.9	5,610	64.6
A 90 - A 93	Influenza, pneumonia, bronchitis, emphysema and asthma	1,651	6.3	2,834	58.3
A 86	Diseases of the arteries, arterioles and capillaries	1,521	5.8	2,171	70.1
	Other causes	4,032	15.5		

Table B

Hospital Morbidity in Ontario

	Disease Category (I COA - 8)	No. of Separations	% of Total	No. of Days	% of Total
	Total	**1,374,716**	**100.0**	**14,267,303**	**100.0**
I	Infective and Parasitic diseases (001-010)	32,017	2.3	262,133	1.8
II	Neoplasms (011-040)	90,950	6.6	1,293,431	9.1
III	Endocrine, Nutritional and Metabolic Diseases (041-048)	25,555	1.9	372,098	2.6
IV	Diseases of the Blood and Blood Forming Organs (049-051)	9,074	0.7	89,955	0.6
V	Mental Diseases (052-060)	57,410	4.2	1,020,958	7.2
VI	Diseases of the Nervous System and Sense Organs (061-075)	60,798	4.4	852,829	5.9
VII	Diseases of the Circulatory System (076-091)	152,191	11.1	3,272,054	22.9
VIII	Diseases of the Respiratory System (092-103)	143,292	10.4	981,176	6.8
IX	Diseases of the Digestive System (104-121)	148,778	10.8	1,292,063	9.1
X	Diseases of the G-U System (122-135)	149,598	10.8	830,456	5.8
XI	Complications of Pregnancy, Childbirth, Peuperion (136-145)	179,514	13.1	928,500	6.5
XII	Diseases of the Skin and Subcutaneous Tissue (146-148)	21,400	1.5	193,546	1.4
XIII	Diseases of the Musculoskeletal System, Connective Tissue (149-156)	68,460	4.9	881,089	6.2
XIV	Congenital Anomalies (157-163)	14,325	1.0	140,252	0.9
XVI	Symptoms and Ill-Defined Conditions (169-170)	73,285	5.3	360,350	2.5
XVII	Accidents, Poisonings and Violence (171-185)	118,782	8.6	1,377,356	9.6
XVIII	Supplementary Classifications (186-188)	29,287	2.1	119,477	0.8

Source: Statistics Canada, Hospital Morbidity, 1976. Catalogue 82-206, Statistics Canada, Ministry of Supply and Services, Ottawa, Canada, 1979.

APPENDIX 2

COMPENSATED INJURIES - OTHER JURISDICTIONS

Table A

Compensated Occupational Disease Cases by Category of Disease (U.S.)[a,b,c]

Disease	% of Total Disease Cases Compensated
Skin Diseases	23.1
Dust Diseases	16.2
Respiratory Conditions Due to Toxic Agents	11.6
Poisoning	4.7
Disorders Due to Physical Agents	1.0
Disorders Due to Repeated Trauma	33.0
Cancer and Tumours	1.0
Other	9.5
Total	**100 %**

a Distribution of occupational disease cases in a closed-case survey of 1,052 cases. Barth, P.S. and Hunt, H.A. Workers' Compensation and Work-Related Illnesses and Diseases, M.I.T. Press, 1980.

b Barth and Hunt caution that this survey may not be representative. Specifically, whereas this survey indicated that 16.2% of occupational disease cases were dust diseases, OSHA reported that 0.9% of lost workday occupational disease cases involved dust diseases. Possible explanations for this discrepancy are discussed in their text.

c A review of annual reports revealed that disease cases are approximately 1 or 2% of all cases in the U.S. as well.

Table B

Spells of "Certified Incapacity" (U.K.) by Disease[a]

Prescribed diseases = 2.3% of accidents

Disease	% of Total Disease
Lead Poisoning	0.3
Squamous Cell Skin Cancer	0.1
Bursitis or Subcutaneous Cellulitis (hand, knee or elbow)	8.3
Traumatic Inflammation (tendons of hand or forearm)	24.0
Tuberculosis	0.4
Inflammation or Ulceration (mucous membrane of mouth or upper respiratory passages due to dust, liquid or vapour)	0.3
Non-infective Dermatitis of External Origin (excluding that due to ionizing particles or electromagnetic radiation)	65.7
Other Diseases	1.0

Notes

a Adapted from Barth and Hunt, 1980, op. cit. from Social Security Statistics, 1975, Department of Health and Social Security, U.K., 1977.

Table C

Cases Accepted for Reparations by Cause (Belgium)[a]

Total = 5,737 cases

Cause of Disease	% of Total Diseases
Chemical agents	3.2
Skin diseases caused by chemicals (not otherwise shown)	2.9
Silicosis	0.9
Inhalation of substances (not otherwise shown)	89.6
Infections or parasites	2.6
Physical agents	1.5
	100%

a Adapted from data in Barth and Hunt, op. cit., from Rapport Annual, Exercise 1974.

Table D

Occupational Diseases and Occupational Injuries (Switzerland)[a,b]

Accidents	1,346,574	
Diseases	23,856	(1.7%)
Silicosis	1,593	(0.1%)

a Adapted from data in Barth and Hunt, op. cit. from Resultats de la Statistique des Accidents.

b Data for 1968-1972.

APPENDIX 3

Schedule III - Ontario Workers' Compensation Act

Column 1	Column 2
Description of Disease	Process
1 Anthrax	Handling of wool, hair bristles, hides and skins
2 Infected blisters	Any process involving continuous friction
3 Bursitis	
4 Epitheliomatous cancer or ulceration of the skin due to tar, pitch, bitumen, mineral oil or paraffin or any compound, product or residue of any of these substances	Handling or use of tar, pitch, bitumen, mineral oil or paraffin or any compound, product or residue of any of these substances
5 Compressed-air illness or caisson disease	Any process carried on in compressed air
6 Dermatitis venenata	
7 Poisoning and its sequelae by	
i arsenic	Any process involving the use of arsenic or its preparations or compounds
ii benzol	Any process involving the use of benzol
iii beryllium	Any process involving the use of beryllium or its preparations or compounds
iv. brass, nickel or zinc	Any process involving the use of brass or nickel or melting or smelting zinc
v cadmium	Any process involving the use of cadmium or its preparations or compounds
vi carbon bisulphide	Any process involving the use of carbon bisulphide or its preparations or compounds
vii. carbon dioxide	Any process involving the evolution of carbon dioxide
viii. carbon monoxide	Any process involving the evolution of carbon monoxide
ix chlorinated hydrocarbons (carbon tetrachloride, trichlorethylene, tetrachlorethane, trichlornaphthalene and others)	Any process in the manufacture or involving the use of these substances
x. chrome	Any process involving the use of chromium or its compounds
xi. lead	Any process involving the use of lead or its preparations or compounds
xii. mercury	Any process involving the use of mercury or its preparations or compounds
xiii nitro derivatives and amino derivatives of benzene, phenol and their homologues (trinitrotoluene, dinitrophenol, anilin and others)	Handling any nitro derivatives or amino derivates of benzene or phenol or any of their homologues or any process in the manufacture or involving the use thereof
xiv. nitrous fumes	Any process in which nitrous fumes are evolved
xv phosphorus	Any process involving the use of phosphorus or its preparations or compounds
8 The pneumoconioses other than silicosis	
9. Any disease due to exposure to X-rays, radium or other radioactive substances	
10 Respiratory disease due to the inhalation of materials used in non-offset sprays	Any process or occupation involving the use of non-offset sprays in the printing industry
11 Retinitis due to electro-welding or acetylene-welding	
12 Silicosis	Mining or quarrying, cutting, crushing, grinding or polishing stone, or grinding or polishing metal
13 Teno-synovitis	
14 Tuberculosis contracted by an employee employed by and in,	
i. a hospital, jail, sanatorium, convalescent home, nursing home home for the aged, health unit or visiting nursing association to which Part I of the Act applies, or	
ii. a laboratory, reform institution, health unit or treatment centre operated by the Province of Ontario	
15. Ulceration of the corneal surface of the eye, due to tar, pitch, bitumen, mineral oil or paraffin, or any compound, product or residue of any of these substances	Handling or use of tar, pitch, bitumen, mineral oil or paraffin, or any compound, product or residue of any of these substances

APPENDIX 4

Occupational Factors in Degenerative Disc Disease

Back pain is one of the major causes of disability in our society. Studies have shown that back problems cause from 4% to 20% of all sickness absences,[1] and are second only to upper respiratory tract illness as the cause of time lost due to illness.[2] Moreover, studies have shown that 25% of all working men are affected each year[3] and that one of every 25 men change jobs because of back pain.[4] According to experts in the field, degeneration of the intervertebral disc accounts for at least 70% of low back disability in industry.[5]

While degenerative disc disease (DDD) is not characteristic of any particular trade or occupation, it is now well-recognized that occupational as well as non-occupational factors contribute to its development.[6] As a disease of wear and tear, it has often been associated with the aging process itself. One study found autopsy evidence of degeneration of the spine in 80% of males at age 49 and 60% of females of corresponding age. By the age of 80, some evidence of DDD was found in all cadavers.[7] However, some experts argue that DDD is not an inevitable accompaniment of aging.[8] Some authors have found that although certain x-ray changes (spurring of the vertebral body) show a direct relationship to aging, the degenerative changes of the intervertebral discs, as measured by radiological narrowing, are not an accompaniment of aging itself, but do in fact, correlate with a history of injuries.[9] Hereditary factors have been suggested,[10] and many studies have revealed the role of various workplace factors. Anderson recently reviewed the extensive data implicating the role of physically heavy work, static work postures, frequent bending and twisting, lifting and forceful movements, repetitive work and vibration in the development of low-back pain in industry.[11] A brief look at the pathophysiology (how the condition develops) will help explain how these factors contribute to the development of this disease.

The intervertebral discs, important in stabilizing the spine, begin to degenerate as early as the second decade of life through progressive scarring of the blood vessels to the disc. This gradually leads to total disappearance of blood flow.[12] The disc must then receive its nutrition through a much less efficient means, diffusion through the annulus fibrosus, (the outer ring of the disc) and the contaginous vertebral end plates.[13] The disc is thereby rendered essentially without regenerative capacity. As a person ages, biochemical changes take place in the disc (water and total sulfate ester contents fall while the glycoproteins and noncollagenous proteins increase, resulting in increased precipitation of collagen).[14] This results in loss of disc height, which in turn causes the vertebral bodies to come closer together, requiring adaptive changes in the surrounding joints and ligaments. Secondary degenerative processes may then occur in the spine which may be manifested by pain and disability. It has been pointed out that the appearance of symptoms may seem to be quite arbitrary and may often appear even before the degenerative changes are detectable on x-ray.[15]

It has been unequivocally shown that mechanical strain may cause degeneration of the spine. Studies have shown that continuous slight pressure on the disc may cause its degeneration with rupture of the annulus fibrosus and it is considered probable that degeneration of the spine is at least hastened by a series of slight injuries which ultimately cause minor irreversible damage.[16] Some studies have shown that repeated compressive stresses and lifting in particular, can cause microfractures in the cartilaginous end plates which are believed to thereby alter the fluid transfer to the disc.[17] Biochemical studies have indeed shown that shearing mechanical force can cause alteration in the colloidal state of proteins to promote their denaturation.[18] Chovin studied the stresses induced at the various joints of the lower spine during weight lifting at various body postures. He showed how the skeletal muscles positioned to exert high forces at these joints to counteract the forques created by the load, are the primary source of compression forces in the lumbosacral discs.[19]

Wickstrom reviewed both epidemiological and animal studies on the various occupational factors promoting the development of degenerative back diseases.[20] He showed that the results obtained in a very thorough study conducted by Hult[21] revealed that severe or repetitive symptoms and the pronounced signs of DDD were two to three times more common among the heavy workers than among the workers in physically light occupations. Separate investigations of different occupational groups have claimed that miners,[22] foundry workers,[23] metal-industry workers,[24] forest workers,[25] truck drivers,[26] and certain hospital workers involved in occupations requiring heavy lifting[27] were among those with increased incidence of DDD. Wickstrom's review also showed that injury, heavy lifting and carrying, different work postures, too rapid a work pace, whole body vibration and unfavourable climate were all probable contributors to DDD.[28]

Studies have specifically shown that low-back pain is a major reason for early retirement among construction workers. Damlund et al., conducted a survey of Danish semi-skilled construction workers and found that 40% reported low-back pain as one of the reasons for retirement, whereas the corresponding percentage was 25 in the reference group. Low-back pain occurred in the year before retirement for 68% of the construction workers and 50% of the referents.[29]

Thus, disability attributable to DDD is a major problem in society. Although it is not particular to any specific occupational group, certain occupational demands are more likely to hasten the disease process. It is hoped that recognition of the contributory factors will promote preventive measures. Despite the complexity of the problem, the workers' compensation system must somehow take up responsibility in this area.

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APPENDIX 5

Statistics Concerning Claims for Noise-Induced Hearing Loss

Table A

Total Allowed and Rejected Claims

Year	Total Claims Received[a]	Number Allowed[b]	% Allowed	PD Awards[b] Granted	% of Total
1978	2,038	1,379	67.7	1,338	65.6
1979	1,925	1,305	67.8	1,045	54.2
1980	2,477	1,432	57.8	950	38.3

a Source: Program Planning and Statistical Services
b Source: Dr. Wm. J. McCracken, Executive Director, Medical Services Division

Table B

Claims Included in Survey of Industrial Diseases Peformed for This Inquiry[a], by Claim Status.

	Claims	Percent of Total	Percent of the Adjudicated[b]
Allowed[c]	98	64.5	71
Rejected	40	26.3	29
Claimant did not reply[d]	12	7.9	-
No action[e]	2	1.3	-
Total	**152**	**100.0%**	**100.0%**

a Claims set up prior to July 1, 1979 (selected backwards from date)
b Excludes d and e
c Allowed for compensation or medical aid: 65 claims (42.8%) received permanent disability awards
d Claimant failed to reply to request for evidence and claim was dropped
e Claim discontinued because of incomplete information.

Table C

Frequency Distribution of Claims Received for Industrial Noise Deafness by Weeks from Date Claim Set Up to Date Initial Decision Reached and First Claim Status

Calendar Weeks to Initial Decision	Allowed Medical Aid Only Claims	Allowed Compensation and Medical Aid Claims	Allowed Death Benefit Claims	Rejected Claims	Claimant Did Not Reply Claims	No Action Claims	Total Claims	Cumulative %
0	-	-	-	-	-	-	-	0.0
0 to 1	-	-	-	-	-	-	-	0.0
1 to 2	-	-	-	-	-	-	-	0.0
2 to 3	-	1	-	-	-	-	1	0.7
3 to 4	-	-	-	-	-	-	-	0.7
4 to 5	1	2	-	1	-	-	4	3.3
5 to 6	3	-	-	1	-	-	4	5.9
6 to 7	-	1	-	1	1	-	3	7.9
7 to 8	-	2	-	2	-	-	4	10.5
8 to 9	1	-	-	6	2	-	9	16.4
9 to 10	3	2	-	-	-	1	6	20.4
10 to 11	2	1	-	2	1	-	6	24.4
11 to 12	-	1	-	3	-	-	4	27.0
12 to 13	3	3	-	1	3	-	10	33.6
13 to 16	10	17	-	14	6	1	48	65.2
26 to 52	12	15	-	11	4	-	42	92.8
52 to 104	2	5	-	4	-	-	11	100.0
104 to 156	-	-	-	-	-	-	-	100.0
Unknown	-	-	-	-	-	-	-	100.0
Total	**37**	**50**	-	**46**	**17**	**2**	**152**	
Mean Weeks	23.5	27.3	-	22.2	15.9	24.1		
S.E.M.	2.6	3.1	-	2.0	5.9	1.5		
Standard Deviation	15.8	21.8	-	15.9	8.3	18.0		

This sample study includes only industrial noise deafness claims set up prior to July 1, 1979.

Program Planning & Statistical Services Branch
December 15, 1981

APPENDIX 6

Occupational Stress and Cardiovascular Disease

Cardiovascular disease is the leading cause of death in Ontario. Myocardial infarction, often called "heart attack" occurs when the blood flow to the heart is inadequate to meet its needs and results in the death of heart muscle (myocardium). Factors that determine whether or not the infarct will be fatal include the extensiveness of the area of tissue death, the location of the infarct in relation to the electrical conduction system of the heart and the state of health of the remaining myocardium. The imbalance between the needs of the heart muscle for oxygen and the blood supply may result from increased demands by the myocardium or from decreased circulation through the coronary arteries.

The most common factors increasing the demands on the heart are exertion and emotional stress. Various diseases, such as hyperthyroidism, infections, or obesity, or physiological states such as pregnancy, or other factors such as increased temperature which demands an increased heart rate, will increase the heart's need for oxygen. Decreased oxygen supply to the heart generally results from a partial or complete occlusion of coronary arteries, often due to the presence of some formed elements of the blood (coronary thrombosis), usually in association with atherosclerosis, or the fatty deposits in the coronary artery wall attributable to a disorder in the metabolism of lipids. Atherosclerotic heart disease is the most common underlying cause of cardiac death. Other causes of inadequate oxygen supply to the heart include blood loss, anemia, or exposure to various asphyxiants or toxins. Some poisons may also act directly on the heart muscle to decrease its ability to pump.

Angina pectoris is the sensation of pressure, pain or other discomfort in the chest and/or jaws or upper extremities that occurs when blood flow to the heart is insufficient to meets its demands. Unlike an infarct which causes scarring or permanently reduced functioning of the heart, coronary insufficiency is transient.

Although an assessment of the events precipitating a myocardial infarction (or coronary insufficiency for that matter), is indeed important, the following discussion will relate largely to the development of the underlying cardiac disease, and specifically to the role of occupational stress in this regard. Whereas much is known about the pathophysiology of coronary artery disease, there is still a great deal of uncertainty about the relative importance of each of the "risk factors" known to be associated with it. It has been increasingly appreciated that psychological stress is one of the factors that contribute to the development of coronary artery disease, and there are numerous publications specifically implicating work-related stress as a major contributor. A review of the literature in this area is well beyond the scope of this report; a list of publications on psychosocial aspects of work and health from the Institute for Social Research at Ann Arbor, Michigan, runs to 23 pages alone. Cooper and Marshall reviewed the occupational sources of stress relating to coronary heart disease (CHD) and mental illness,[1] and Chadwick recently reviewed the review articles relating psychological job stress to coronary heart disease for NIOSH.[2]

The first notion that emotional stress might be a major cause of coronary heart disease came in the 1860s from Claude Bernard himself.[3] His work on the autonomic nervous system sowed the seeds for the development of psychosomatic medicine, an entire field of medicine dealing with the role of psychological patterns in the development of physical diseases. Objective evidence that there is a strong psychogenic element in the causation of cardiovascular disease awaited the research of Friedman and Rosenman in the 1950s.

Their initial efforts were simple but productive. Through administering questionnaires, they found that 70% of physicians treating patients with coronary disease as well as lay people, thought that the major causal factor was indeed emotional in origin; specifically characterized by excessive "drive." They then designed a carefully controlled study of 40 accountants monitoring weight, diet and exercise, and found that in spite of the constancy of these factors, there were marked elevations of cholesterol level at times of maximum stress.[4] This led to further research and to the delineation of a coronary-provoking Type A behaviour pattern, characterized by striving, hard driving motivation, time urgency and a preoccupation with deadlines. Type B was taken as the converse of Type A.[5]

On the basis of these findings, Friedman and Rosenman began a prospective study of over 3,500 people which has now been running for about 16 years. The latest reports confirmed the importance of such well-recognized risk factors as a family history of coronary disease, elevated blood pressure, cigarette smoking, and high serum concentrations of cholesterol, beta-lipoproteins and neutral fat. Above all, it confirmed the importance of behavioural patterns. Type A individuals were found to have twice the risk of developing CHD as Type B individuals.[6]

Aside from the clinical and epidemiological studies on the relationship between emotional stress and coronary artery disease, there is a vast body of evidence linking both physical and emotional stress with alterations in various biochemical functions of the body. The work of Selye, for example, illustrating the increase of blood sugar, the accelerated heart rate, the increase in blood pressure, and so on, in response to stress is well known.[7] Various European studies, such as that by Carlson *et al.*, have added further knowledge to the biochemical reactions in response to various stimuli.[8] The advent of technological advancements in monitoring equipment has made it possible to obtain considerably detailed physiological information in association with various activities. For example, one study examined the effects of long-distance flying on aircrew and passengers by simultaneously recording electrocardiogram, electroencephalogram, and blood pressure in combination with time, temperature and events.[9] By combining these biochemical and physiological measures of the responses to stress, the authors felt that it was possible to obtain objective evidence relating occupational stress to heart disease.[10]

Occupational stressors include physical conditions such as noise, vibration, temperature extremes, poor lighting and ventilation as well as psychological stressors. (The role of physical and chemical factors in coronary artery disease will be summarized later.) Job dissatisfaction, unclear demands by superiors, lack of job security, shiftwork, compulsory overtime, work overload (too many responsibilities, competing demands, etc.) as well as underload (monotonous, machine-controlled tasks) have all been unequivocally linked to adverse physical and psychological well-being.[11] For example, in French and Caplan's summary of the research on work overload, they suggested that both qualitative and quantitative overload produce at least nine different symptoms of psychological and physical strain: job dissatisfaction, job tension, lower self-esteem, threat, embarrassment, high cholesterol levels, increased heart rate, skin resistance and more smoking.[12] It has been pointed out that four of these (job dissatisfaction, elevated cholesterol, elevated heart rate and smoking) are indeed risk factors in heart disease.[13]

Coburn recently studied over a thousand male workers in Victoria, B.C. in a variety of white- and blue-collar jobs.[14] He found that feelings of discontent or job strain are widespread in the population, with the actual number reflecting the phraseology of the question. His findings, remarkably similar to a parallel study in Portland, Oregon, showed that 18% were dissatisfied with their jobs, 14% said they disliked their jobs, while 47% stated that they would choose a different type of job if they could. 13%

felt their jobs were mostly dull and monotonous or worse, while 32% stated their jobs subjected them to a great deal of stress. Combining responses, he thus showed that 72% found their job to be monotonous, wanted a different type of work or considered their job to be highly stressful. These findings are also apparently similar to those shown in both Canadian[15] and U.S. national samples.[16] Coburn pointed out the relevance of these findings in citing a Swedish study of blue-collar workers which found that the most consistent job characteristics correlating with biochemical parameters of stress (the production of adrenaline and noradrenaline) were those referring to different aspects of monotony and machine control. The worst jobs were those combining heavy work load and routine repetitive work.[17] The list of studies linking various job characteristics with stress responses is too long to review here. In fact, NIOSH has published a variety of ways to conceptualize and analyze work stresses.[18] Indeed, work in Europe on this subject has advanced to the point that psychosocial work standards or goals have been written into legislation in the Scandinavian countries. Coburn's studies led him to the conclusion that work factors explain 7-10% of the variance in psychological well-being and 3-6% of the variance in physical well-being; job factors and job attitudes combined explained 12-15% of the variance in psychological well-being, 6-8% in physical well-being and 12-16% in overall well-being.[19]

In view of the studies suggesting that stress is causally related to coronary heart disease, and that various occupational demands are inducers of stress as well as other factors in psychological and physical malaise, it might be expected that workers exposed to stressors would have a higher incidence of coronary heart disease. Several studies do indeed suggest this finding. In an investigation of mortality rates of men in California, for example, Breslaw and Buell observed that workers in light industry under the age of 45 who were on the job more than 48 hours a week, had twice the risk of death from CHD compared with similar workers working 40 hours or less a week.[20] The prospective findings from the Framingham heart study with respect to women, work and coronary heart disease showed that CHD rates were almost twice as great among women holding clerical jobs as compared to housewives. Moreover, the most significant predictors of CHD among clerical workers were: suppressed hostility, having a nonsupportive boss, and decreased job mobility. Among working women, clerical workers who had children and were married to blue collar workers were at the highest risk of developing CHD.[21]

Those who tend to downplay the role of the workplace and emphasize the individual factors often point out that these stresses are self-imposed, or attributable to the personality of the individual. Rosenman and Friedman actually propose that the major cause of increase in CHD over the century stemmed from the increasing patterns of Type A behaviour by individuals.[22] However, they and many others note that there are strong indications that work environments not only enhance but also reward Type A behaviour patterns.[23] Rosenman's research strongly suggests that when many individuals enter occupations as switchboard operators, taxi cab drivers, assembly line workers, managers and so on, they often do not possess A type patterns of behaviour. However, increased time pressures, demands for speed, and circumstances required by the job can make a relaxed Type B into a Type A, or a less extreme Type A into a more exaggerated Type A.[24] Davidson and Cooper reviewed the literature in this area and concluded that conditions most responsible for facilitating Type A behaviour are those encountered in the work environment.[25] It is also noted that occupational stressors can have similar physiological effects in Type B individuals. Rose et al.,[26] for example, discovered that Type B air-traffic controllers had a higher incidence of hypertension than their Type B colleagues, and blamed the chronic and unpredictable stressors at work for their poor health. Cooper and Marshall summarized the evidence linking occupational sources of stress, individual characteristics and ultimate disease as shown diagrammatically in the bottom portion of Table A.

Table A

Sources of Stress at Work	Individual Characteristics	Symptoms of Occupational Ill Health	Disease
Intrinsic to Job: poor physical working conditions (noise, heat, cold, dust, fumes, crowding, etc.) work overload time pressures physical danger, etc.			
Role in Organization: Role ambiguity Role conflict Responsibility Conflicts re: organizational boundaries, etc.	**The Individual:** Level of anxiety Level of Neuroticism Tolerance for ambiguity Type A behavioural pattern	Diastolic blood pressure Heart rate Smoking Depressive mood Escapist drinking Job Dissatisfaction Reduced aspirations, etc.	Coronary heart disease Mental ill health
Career Development: Overpromotion Underpromoton Lack of job security, etc.			
Relationships at Work: Harrassment Threat of disciplinary actions Poor relations with subordinates, colleagues or supervisior, etc.	**Extra-organizational Sources of Stress:** Financial insecurity Family problems Life crises, etc.		
Organizational Structure: Little or no participation in decision-making Restrictions on behaviour (budgets, etc. Office politics Lack of effecive consultation, etc.			

Source Adapted from Cooper, C.L. and Marshall, J., J.Occ.Psych. 49:11,1976

Finally, it is important to allude to the evidence linking physical hazards in the work environment to cardiovascular risk factors. While there is considerable controversy in this area, various studies have suggested that noise exposure is a source of hypertension,[27] as well as elevated heart rate, and increased CHD.[28] Whole body vibration has also been implicated.[29] In a study of foundry workers in Brazil under the age of 35, continued exposure to intense heat was associated with hypertension.[30] Exposure to nitroglycerin and ethylene glycol dinitrate has been linked to hypertension[31] and to cardiac mortality. Anilines, nitrobenzenes, chloroform, trichloroethylene, azides, carbon disulfide, carbon monoxide, cyanide, ammonia, chlorine, phosgene and chloroformates have been suspected to be linked to cardiovascular disease.[32]

Thus, cardiovascular disease is truly multifactorial in origin. There is, however, reason to believe that occupational factors, particularly occupational stress, may play a significant role in the causation of this devastating killer. The only numerical estimate found in the literature as to the role of occupational factors estimated 5-10% of cardiac deaths are work-related, 25% are due to smoking (note that several studies suggest that smoking itself is work-related), and 50% are related to diet.[33] Rosenman and others purport that behavioural patterns are the major causative factor in CHD, and the research of Cooper, Coburn and others suggests that the workplace is a major source of stress in our society, company doctors readily admit that the purpose of the pre-employment physical examination is to screen out workers whose health problems may be aggravated by the work environment, and studies have shown that the main reason given by employers for avoiding hiring applicants with cardiac disease was fear of occupational claims against the firm[34], little attention is devoted to reducing the workplace factors responsible for this aggravation.

With this as background, Section 3 of Chapter IV will examine the policy and experience of the Ontario Workers' Compensation Board regarding cardiac claims. Suffice it to state that the issue of "occupational stress" is one of the greatest challenges to the very framework of workers' compensation.

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CHAPTER II

CANCER AND THE WORKPLACE

A. ISSUES AND DIMENSIONS OF OCCUPATIONAL CANCER

1. Cancer in Ontario - The Facts and Figures

Cancer is a devastating disease. The toll it takes in human pain and misery, as well as in both direct and indirect costs is beyond estimation. Cancer was directly responsible for 14,252 deaths in Ontario in 1979 alone and contributed to the suffering of yet another 1,839 people who died that year in Ontario[1] (see Table IIA-1). Cancer is the second most common cause of death for all age groups over age 5 and accounts for the demise of almost 20% of all Ontarians[2] (see Appendix 1 in Part B of Chapter I).

As will be discussed in Part B of this chapter, while cancer accounts for only a tiny percentage of total workers' compensation claims, it is the single largest cause of death in all fatal disease claims filed with the Workers' Compensation Board (i.e., excluding industrial accidents). Presently cancer accounts for about 20% of all fatal claims. Thus apart from its devastating effect on the population at large, cancer is a significant cause of concern from the compensation point of view.

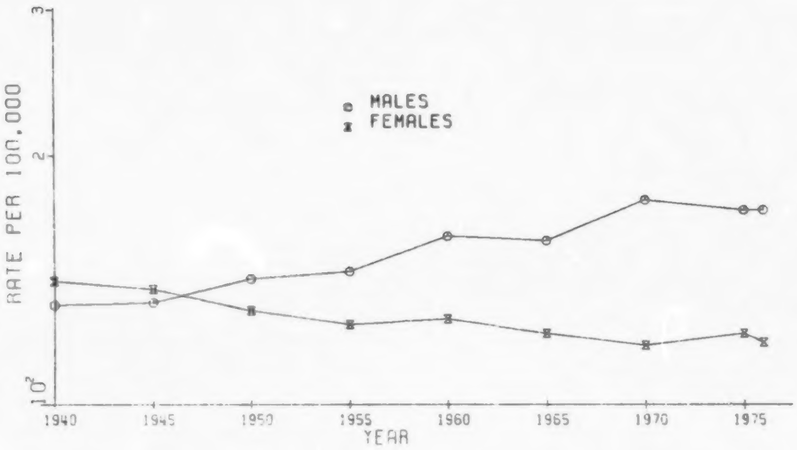
Aside from Quebec which has the highest cancer mortality rate in Canada, Ontario has several areas of exceptionally high (age-standardized) cancer mortality rates - including Metro Toronto, Hamilton-Wentworth, several neighbouring census districts, and several areas in Northern Ontario.[3] When one notes that the various "cancer maps" have shown correlations between various industrial regions and cancer rates,[4] these findings should not be surprising.

While it is widely recognized that cancer is largely a preventable disease[5], the cancer statistics have shown that little overall improvement has occurred over the years. As the population has grown, the total number of people dying of cancer in Ontario has also increased. As well, the percentage of the Ontario population dying of cancer is steadily increasing. In 1976 there were 12,657 deaths from cancer, representing 20.4% of all male deaths and 21.5% of all female deaths. In comparison, the proportion of total deaths from cancer in 1940 was 11.5% and 14.9% in males and females respectively[6] (see Table IIA-2).

It should be noted, however, that cancer mortality rates do indeed increase with age. Whereas at the turn of the century, life expectancy was limited as many people died of infectious diseases, now more people are living long enough to die of cancer. Thus the greater proportion of older-aged individuals in the Ontario population accounts for much of the increase. Nonetheless, even the "age-adjusted" rate shows a slight overall rise. When the changes in the age distribution of the Ontario population between 1940 and 1976 are taken into account, the age-adjusted mortality rates for cancer in females show a 16% decline over this period, whereas males show a 30% increase[7] (see Figure IIA-1).

Fig. IIA-1

Age Adjusted Mortality Rates for all Cancer Sites by Sex, Ontario, 1940–1976*

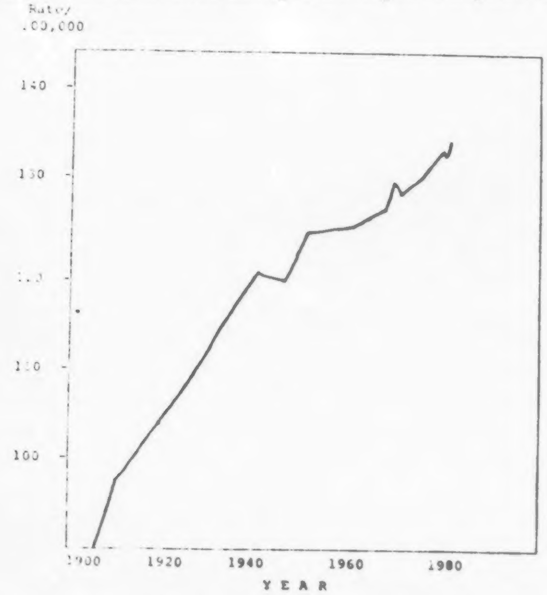


* Age adjusted to the 1941 population of Ontario.

Source: OCTRF Cancer in Ontario, 1978.

Figure IIA-2

Age Adjusted Cancer Mortality Rates per 100,000 U.S. Population(1900–1980)



For age adjustment the 1940 United States population was used as standard.

Source: Davis et al., Banbury Report No. 9, Cold Spring Harbor, 1981.

Table IIA-1

Total Deaths with Mention of Cancer, Ascribed to Other Causes, and Autopsies, Ontario Residents, 1979[1]

Type of Cancer	Total Deaths With Cancer	Deaths Ascribed To Cancer	Deaths Ascribed To Cause Other than Cancer		Autopsies	
			Number	Per Cent	Number	Per Cent
Lip	9	8	1	11.1	0	0.0
Tongue	86	77	9	10.5	14	16.3
Rest of mouth	98	84	14	14.3	10	10.2
Pharynx and tonsil	131	122	9	6.9	22	16.8
Esophagus	299	281	18	6.0	50	16.7
Stomach	810	756	54	6.7	101	12.5
Intestine, except rectum	1,662	1,481	181	10.9	200	12.0
Rectum and anal canal	618	541	77	12.5	68	11.0
Other digestive system	1,536	1,424	112	7.3	292	19.0
Nose, middle ear and accessory sinuses	22	20	2	9.1	3	13.6
Larynx	141	118	23	16.3	26	18.4
Lung, bronchus and trachea	3,397	3,176	221	6.5	608	17.9
Other respiratory	33	29	4	12.1	15	45.5
Bone	51	47	4	7.8	10	19.6
Malignant melanoma	139	127	12	8.6	36	25.9
Other cancer of skin	67	39	28	41.8	4	6.0
Breast	1,525	1,309	216	14.2	231	15.1
Cervix uteri	227	207	20	8.8	33	14.5
Other uterus	224	189	35	15.6	32	14.3
Ovary, tubes, ligament	464	444	20	4.3	76	16.4
Other female genital organs	44	37	7	15.9	3	6.8
Prostate	1,000	708	292	29.2	89	8.9
Testis	15	12	3	20.0	5	33.3
Other male genital organs	13	8	5	38.5	4	30.8
Bladder	461	368	93	20.2	67	14.5
Kidney, other urinary	314	279	35	11.1	72	22.9
Brain and nervous system	382	368	14	3.7	72	18.8
Thyroid gland	41	37	4	9.8	13	31.7
Lymphosarcoma and reticulum-cell sarcoma	163	140	23	14.1	57	35.0
Hodgkin's disease	82	74	8	9.8	24	29.3
Leukemia	694	575	119	17.1	170	24.5
Other sites	1,343	1,167	176	13.1	292	21.7
Total	**16,091**	**14,252**	**1,839**	**11.4**	**2,699**	**16.8**

[1] Data are taken from transcripts of registrations with mention of cancer on the medical certificate and do not agree exactly with the final published figures.

Source: OCTRF Cancer in Ontario, p. 47, 1980

Table IIA-2

Cancer Mortality Rates and Cancer Deaths as a Proportion of Total Deaths by Sex, Ontario, 1940-1976

	MALES					FEMALES				
Year	Total Deaths All Causes	Cancer Deaths	Per Cent of Total Deaths	Death Rate Crude[1]	Death Rate Adjusted[2]	Total Deaths All Causes	Cancer Deaths	Per Cent of Total Deaths	Death Rates Crude[1]	Death Rates Adjusted[2]
1940	20,923	2,413	11.5	127	132	17,580	2,624	14.9	142	141
1945	21,563	2,669	12.4	132	133	17,936	2,864	16.0	144	138
1950	24,502	3,221	13.1	143	142	19,446	3,096	15.9	139	130
1955	25,890	3,689	14.2	139	145	19,544	3,413	17.5	131	125
1960	29,408	4,467	15.2	146	160	22,076	3,948	17.9	131	127
1965	31,275	4,920	15.7	146	158	23,071	4,362	18.9	130	122
1970	32,520	6,080	18.7	160	177	24,249	4,858	20.0	127	118
1975	34,107	6,755	19.8	165	172	26,497	5,607	21.2	136	122
1976	34,225	6,980	20.4	170	172	26,420	5,677	21.5	136	119

[1] Rate per 100,000 estimated population by sex, all ages.

[2] Rate per 100,000 population, adjusted for age, based on the 1941 population of Ontario.

Source: OCTRF Cancer in Ontario p. 49, 1978.

Thus the age-adjusted mortality rate for all cancer (as well as the crude, or unadjusted cancer mortality rate) is on the increase in Ontario. This is consistent with statistics for Canada as a whole[8] as well as those for the United States[9] (Figure IIA-2).

Doll and Peto point out that improvements in diagnosis and reporting of cancer as well as the decrease in deaths attributable to other causes are among the factors that contribute to this relative increase in cancer.[10] Moreover, it has been pointed out that the increasing rate of cancer is largely accounted for by lung cancer alone. It is, in fact, true that the age-adjusted mortality rates for cancer of the prostate, intestine and bladder in Ontario males appear to have changed little between 1931 and 1975; the mortality rate from stomach cancer has shown a 58% decrease, whereas lung cancer mortality rates in men have increased 14-fold over this period (See Figure IIA-3a). Similarly, cancer of the uterus has shown a 38% decrease while lung, bronchial and tracheal cancer mortality rates for Ontario females have shown a five-fold increase (Figure IIA-3b).[11] The most recent Ontario mortality figures show, in fact, that when lung cancer is removed from cancer mortality rates, the rate for all cancers minus lung cancer actually shows a decline.[12]

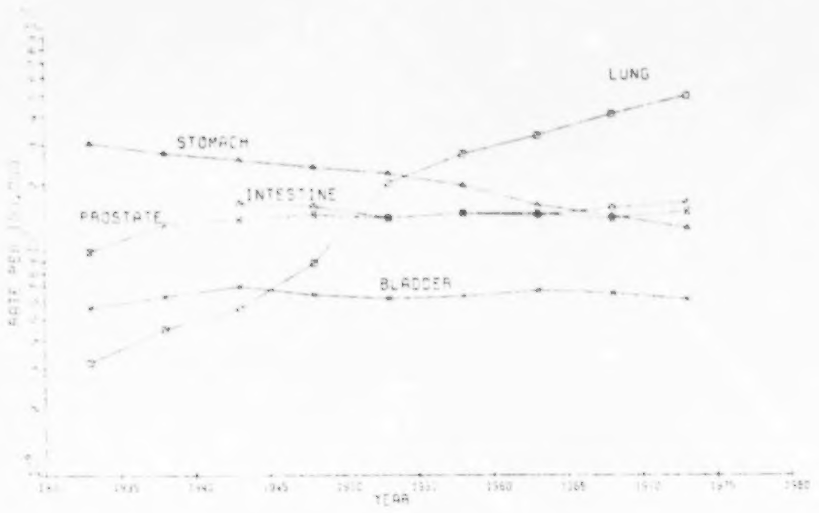
Since mortality data only include those people who die from their disease, and since death certificate data are subject to considerable inaccuracy[13], some people prefer to use incidence data (number of new cases). Age-adjusted cancer incidence rates for 1966 and 1971 were compiled by the Ontario Cancer Research Foundation for selected sites[14] and also reveal an increasing cancer incidence rate here in Ontario (see Table IIA-3). Epstein stresses that the overall increase in cancer incidence rates is even more marked than the increase in mortality rates over the past decade, involving a wide range of organs besides the lung (see Table IIA-4). Moreover, he shows that the increase in cancer incidence for all sites is comparable with that when lung is excluded (see Table IIA-5).[15] (Doll and Peto, however, dismiss these findings, pointing out that trends in the reporting of cancer cases tend to fluctuate considerably, making incidence data even less reliable than mortality data.)[16]

Cancer is horrendous also in terms of its abysmal prognosis (or outlook). It is abundantly obvious from the data (Table IIA-1 for example) that the great majority of people who develop cancer die of their disease. The 5-year survival rates for most cancers have shown little improvement and are as discouraging as ever (See Table IIA-6). It is thus clear that attempts at earlier detection and improved treatment methods have, with few exceptions, failed to stop the scourge of cancer. For lung cancer, killer of some 5,000 in Ontario last year, elaborate efforts using chest x-ray screening and sputum cytology are still being considered, but have so far failed to alter the outcome. As few as 8% of males and 13% of females live 5 years from the time their lung cancer is diagnosed.[17]

Suffice it to state that cancer as a whole is on the increase in Ontario as elsewhere, even when adjusted for the changing age-structure of the population. It is true that a large part of this increase is due to respiratory cancer, and when removed, cancer mortality does begin to show a slight downward trend. Cancer incidence (the biases and uncertainties notwithstanding) still appears to be on the increase even when respiratory cancer is discounted. Even if the current situation cannot be characterized as a "cancer epidemic", the mammoth dimensions of the cancer problem, the pain and suffering it causes and the very poor prognosis of cancer patients is a devastating reality.

Fig. IIA-3a

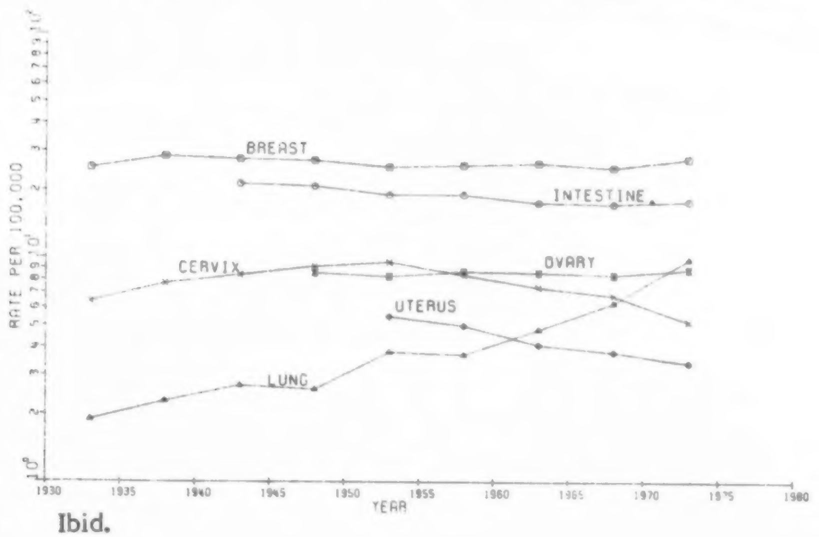
Age Adjusted Mortality Rates for Selected Male Cancer Sites, Ontario, 1931-1975
(Age adjusted to the 1961 male population of Ontario)



Source: OCTRF Cancer in Ontario, 1978.

Fig. IIA-3b

Age Adjusted Mortality Rates for Selected Female Cancer Sites, Ontario, 1931-1975.
(Age adjusted to the 1961 female population of Ontario)



Source: Ibid.

Table IIA-3

Age Adjusted Incidence Rates for Selected Cancers by Sex, Ontario, 1966 and 1971*.

Cancers and Cancer Sites	Males 1966	Males 1971	Females 1966	Females 1971
Stomach	18.6	16.7	11.0	10.6
Intestine except rectum	24.7	25.2	30.4	30.6
Rectum and anal canal	17.5	16.9	12.9	11.3
Larynx	5.4	6.1	0.6	0.8
Lung, bronchus and trachea	45.6	57.9	7.6	11.0
Breast	0.7	0.6	69.2	74.5
Cervix uteri			24.1	19.9
Other uterus			16.1	17.4
Ovary, tubes and ligaments			12.3	11.8
Prostate	38.5	37.4		
Bladder and other urinary	17.9	19.4	6.9	7.3
Hodgkin's disease	3.3	3.2	2.5	2.6
Leukemias	10.4	10.6	7.7	7.0

* Rates per 100,000 population and age adjusted to the 1971 population of Ontario.
Source: OCTRF, 1978, op cit.

Table IIA-4
Average Per Cent Change in Age-adjusted Mortality and Cancer Incidence Rates for selected sites by sex and year, 1969-76, (white population only)

		Average % Change in Mortality Rates 1969-76		Average % Change in Incidence Rates 1969-76	
Site	Sex	Annual	7-Year	Annual	7-Year
All Sites	M	0.9	7.8	1.3	7.9
	F	0.5	3.7	2.0	10.9
Stomach	M	-2.9	-17.4	-2.3	-18.2
	F	-3.6	-22.6	-3.7	-21.1
Colon	M	1.3	10.7	1.5	7.0
	F	0.0	1.9	0.7	2.6
Rectum	M	-3.0	-18.8	1.3	10.9
	F	-3.1	-17.9	1.2	2.7
Pancreas	M	0.0	0.0	-0.5	-5.0
	F	0.3	3.0	0.9	6.7
Lung	M	2.6	21.3	1.4	10.2
	F	7.6	74.5	8.6	78.2
Melanoma	M	4.0	30.0	6.8	54.5
	F	0.8	14.3	6.2	48.8
Breast	F	0.39	3.8	1.8	13.0
Cervix	F	-4.9	-29.1	-5.9	-33.8
Uterus	F	-1.7	-10.5	5.9	38.1
Ovary	F	1.2	8.7	-0.4	8.7
Prostate	M	0.6	5.6	2.3	16.3
Bladder	M	-1.4	-2.8	2.3	10.9
	F			2.5	15.9
Kidney	M	0.6	4.7	1.2	6.7
	F	0.7	5.0	1.3	11.6
Leukemia	M	-0.4	-2.1	-0.2	-0.8
	F	-1.7	-8.8	-1.0	-11.3

The 1970 population was used as standard for age-adjustment.
Source: Epstein, Nature 289: 5794, 1981, op. cit.

Table IIA-5

Changes in U.S. Cancer Incidence Rates from 1970 to 1975

	Average % increase in incidence rates, 1970-75			
	Cancers of all sites		Cancers of all sites except lung	
Groups	Annual	5-Year	Annual	5-Year
White male	0.9	4.7	0.9	4.6
Non-white male	2.3	11.9	2.7	14.3
White female	2.2	11.6	1.8	10.2
Non-white female	6.1	34.6	5.7	32.2

Source: Epstein, 1981, op cit.

Table IIA-6

5-Year Survival Rates[a] for Major Cancer Sites at All Stages, by Sex, 1965-1969[b]

Sites	Male (%)	Female (%)
Lip	84	85
Tongue	32	Insufficient Data
Mouth	45	56
Pharynx	21	30
Esophagus	3	6
Stomach	12	14
Colon and Rectum	44	46
Liver	6	9
Pancreas	1	2
Larynx	63	59
Lung	8	13
Breast	Insufficient Data	65
Uterus	Not Applicable	66
Ovary	Not Applicable	34
Prostate	57	Not Applicable
Kidney	41	43
Bladder	62	62
Melanoma	61	75
Brain	25	33
Thyroid	80	87
Lymphosarcoma	34	37
Reticulosarcoma	18	19
Hodgkin's Disease	53	57
Multiple Myeloma	17	16
Leukemia (Total)	14	15

Notes:

[a] adjusted for normal life expectancy

[b] Source: Ca-A Cancer J. for Clinician 29, 1: 21, 1979

Thus it is not surprising that cancer is as much a focus of concern in Ontario as it is elsewhere. Those who believe that industrial carcinogens play a significant role in cancer causation are passionate and unrelenting in their efforts to seek enactment of regulations to prevent occupational cancer, and in their quest for redress for those unfortunate cancer victims and their families whose suffering may be linked to factors in the workplace. Although presently only a small fraction of cancer cases in Ontario reach the Workers' Compensation Board, the difficulties in determining the work-relatedness of any particular case have resulted in a higher rejection rate for cancer claims than almost any other category of claim and the rejection rate for cancer claims is more than ten times higher than that for claims overall. (This will be discussed at length in Part B of this chapter). Cancer has thus already been the focus of controversy for some time and promises to be an area of major concern in the workers' compensation system in years to come.

2. Current Knowledge about Cancer Causation

2.1 The Nature of Cancer and Carcinogenesis

Cancer, which comes from the Latin word for "crab", refers to a malignant tumour or neoplasm. While cancer is not a single disease but rather a group of diseases, it has been observed that cancer cells all share certain characteristics. They are irreversibly altered cells which possess new properties not shared by normal cells; in their malignant form, they are harmful to the rest of the body; they develop independently of the cells of origin, and their progressive growth is not subject to growth equilibrium as in normal tissues.[18]

The key to the harm caused by cancer and what distinguishes malignant from benign tumours, is the rapid and disorganized pattern of growth of the former. The invasion of surrounding tissues by malignant neoplasms is a highly destructive process causing severe damage to the invaded tissue preventing it from performing its normal functions. The cancer may also fail to develop a blood supply sufficient to nourish its rapid growth and therefore will resort to parasitizing on the host tissues. As the blood supply may not be adequate to support both the cancer and the normal tissue, death of cells may occur. The dead cells may then be the focus of bacterial invasion and further destruction, resulting in the production of harmful substances which add to the debilitation of the body as a whole.

Apart from infiltrating surrounding tissues, malignant neoplasms spread by a process of seeding. Small clumps of cancer cells may be transported to distant organs to produce secondary deposits (or metastases) which then continue to perpetuate the neoplastic process and seed further metastases. The metastases often do more harm than the primary tumours. Generally the spread takes place via the bloodstream or the lymphatic system or by transcoelomic spread, or spread across a space within the abdomen or chest. (Metastasis through the bloodstream takes place when invading cancer cells become trapped in the capillary networks, such as those in the lungs and liver. Lymphatic spread occurs by a similar process. Small clumps break off and are carried in the lymphatic channels to adjacent lymph nodes where they give rise to secondary malignant growth which may take over the lymph nodes and result in further spread. Transcoelomic spread takes place when a malignant tumour has infiltrated all the way through the surface lining and continues to grow within the cavity either in clumps or as a continuous sheet of malignant cells.)

The secondary neoplasms, formed by metastasis, have the characteristics of the primary neoplasm, and certain patterns of metastasis commonly occur based in part on the direction of blood supply and lymphatic drainage. Cancer of the lung, for example, commonly metastasizes to the kidney, liver and brain. As malignant neoplasms may bear little resemblance to the cells of the tissue of origin, determination of site of origin may be difficult. As will be illustrated in Part B of this chapter, this creates problems in determining the work-relatedness of a cancer. For example, in one case presented below, one pathologist felt that the primary tumour was mesothelioma, while a second pathologist concluded that the site of origin was the pancreas. The Board accepted the opinion of the second pathologist and this led to a negative outcome in the case.

Although cancer can arise from any organ of the body, some sites are affected more than others (as shown in Table IIA-7). The ten billion (10^{13}) cells which make up the various tissues and organs of the body are each programmed to carry out their appropriate functions. Some cells divide and multiply continuously during adult life, e.g. skin, lining of intestines and bone marrow cells; some cells seldom multiply or do so only when tissue damage occurs, e.g. the liver and other glands, and some cells do not multiply at all in the adult, e.g. nerve cells. The cells which multiply continuously are the most common sites of cancer, while those cells that multiply only in youth give rise to cancers found mostly in the young. Cancer cells develop from single cells which somehow became free of the normal control of their multiplication, to reproduce similar cells which continue to multiply and which can spread. The variable tendencies to replicate possessed by different cell types account in part for their different susceptibility to mutations and consequently to develop cancer.

In the 1950's and 60's there was a great increase in the knowledge of molecular biology which significantly contributed to the understanding of the mechanisms of carcinogenesis. Basically, cell behaviour is determined by proteins manufactured and controlled within the cell. Each cell inherits its instructions on protein synthesis from DNA (dioxyribonucleic acid) found in the nucleus of the cell. The DNA in turn is made up of four basic building blocks strung together as a double-stranded helix. The specific genetic information of DNA is determined by the sequence of these four bases. RNA (ribonucleic acid) acts as a messenger molecule in the replication process. The expression of the genetic information is controlled by special proteins which interact with the DNA to prevent or stimulate gene expression.

It is now believed that changes in the genetic material of the cell are necessary for the transformation from normal to neoplastic cells.[19] Physical agents, such as x-rays, and many chemical agents are capable of interacting with DNA to produce mutations in the genetic structure of DNA. (Some chemical carcinogens have been shown to react with DNA, and possibly RNA or protein, due to the fact that their various chemical formulae share the feature of being strongly electrophilic[20] - i.e., are either uncharged or positively charged electron-deficient atoms capable of reacting with the nucleophilic, i.e. electron seeking sites in the target

Table IIA-7

Estimated Proportion of Cancer Incidence by Site and Sex[a,b]

Male		Female	
Skin	2%	Skin	2%
Oral	5%	Oral	2%
Lung	22%	Breast	27%
Colon & Rectum	14%	Lung	8%
Pancreas	3%	Colon & Rectum	15%
Prostate	17%	Pancreas	3%
Urinary	10%	Ovary	4%
Leukemia & Lymphomas	8%	Uterus	13%
All Others	19%	Urinary	4%
		Leukemia & Lymphomas	7%
		All Others	15%

Notes

[a] Source: Ca-A Cancer Journal for Clinicians, American Cancer Society 29:1, 1979 data from NCI's Surveillance, Epidemiology and End Results (SEER) Program (1973).

[b] Excludes non-melanoma skin cancer and carcinoma in situ.

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cell, such as the DNA molecule. They thereby cause errors in the genetic coding and are replicated erroneously.)

If agents cause damage to the DNA in the somatic cells in the adult body, they may give rise to cancer and are therefore considered carcinogenic. Damage to DNA in germ cells may raise the level of deleterious mutations for future generations. These agents are known as mutagens. If the mutagenic agents interact with DNA in the somatic cells of the fetus, they may produce developmental errors which appear as birth defects.

2.2 Agents Capable of Causing Cancer

Although the understanding of the factors involved in cancer causation is still incomplete, a great deal of knowledge has been accumulated on substances which can cause cancer and the mechanisms by which they do so.

Studies in experimental animals and observations on human populations have demonstrated several classes of carcinogenic agents. The ability of certain chemicals, of ultra-violet light and of ionizing radiation to cause cancer has been well-documented for many years. The possible role of viruses as etiological agents for human cancer is not clear, but has been shown not to be of significance in the vast majority of cancers.[21]

The importance of chemical carcinogens has been recognized for over two centuries. Exposure in the workplace to soot was first recognized as being linked to cancer when, in 1775, Pott noted that chimney sweeps had a high incidence of scrotal cancer. Occupational exposure to a wide variety of polycyclic aromatic hydrocarbons, the products of incomplete combustion, has been shown to induce tumours in a wide variety of tissues. Coal tar, soot, and certain mineral oils, often used as cutting oils in lubricating, have been implicated. An increased incidence of cancer of the bladder in association with certain aromatic amines was first recognized in 1895 when Rehn noted the high incidence of bladder cancer in workers in the dyestuff industry. Now strong epidemiological data exist implicating benzidine, alpha and beta-naphthylamine and 4-aminobiphenyl, to which rubber workers are exposed. Industrial exposure to alkylating agents such as bis-chloromethylether, an intermediate in the production of ion exchange resins, and inorganic substances such as chromium, nickel, arsenic and asbestos are now all well documented carcinogens. It is abundantly clear that occupational cancer is not a new phenomenon.[22]

Medicinal exposure to chemical carcinogens has also been documented. For example, there is a well established link between in utero exposure to diethylstilbestrol and vaginal cancer. General societal exposures, such as the contamination of food by aflatoxin B, have been implicated in cancer causation, and most notable, is the link between cigarette smoking and cancer. (Appendix 1 contains a list of some of the known human carcinogens, established sites of cancer associated with them and sources of exposure.)[23]

Within the different chemical groupings of carcinogens there are strong structural similarities, but there is little or no apparent similarity between groups of chemicals. Actually, when cancer was first induced in experimental animals in 1915 by applying coal tar to the ears of rabbits, the compounds isolated from the coal tar were found to be rather unreactive and did not prove to be fruitful mutagens. It was eventually learned that these compounds had to be metabolically activated or converted in the body into reactive carcinogenic compounds. Thus the chemical carcinogens were able to be roughly divided into 2 groups on biochemical grounds.[24] The first group are the directly acting substances which do not need metabolic transformation because they are already chemically active. (This group includes the epoxides, lactones, sulfur and nitrogen mustards, halo ethers and acylating agents.) The second group of carcinogens are indirectly acting, i.e. are not capable of acting on the cellular level without some metabolic or chemical activation. (These include aromatic amines, nitrosamines, aromatic hydrocarbons and halogenated aliphatic hydrocarbons such as carbon tetrachloride and vinyl chloride.) These are converted in the body to the "ultimate carcinogen" that is actually responsible for inducing the tumour.

The majority of known carcinogens act indirectly. This leads to complications in terms of species and organ specificity due to differences in enzyme systems and other factors which may influence known or suspected metabolic pathways. (The issue of extrapolating data from animal studies will be discussed below.) The balance between conversion to active and inactive metabolites is believed by some to be the critical factor in the carcinogenic potential of chemicals.[25]

Overall, most tumours seem to be initiated through one or more changes in the informational content of the DNA. However, other data suggest that some cancer cells may arise from epigenetic changes, i.e., changes outside the genetic material. For example, the basis for the carcinogenicity of asbestos is still obscure. At one time it was thought that its chemical constitutents were key; more recently, the physical properties of asbestos fibres have been thought to be of significance. Agents of this nature would not be easily detected by short-term laboratory studies which rely on detecting mutagenic changes in micro organisms.

Electromagnetic radiation of certain frequencies (x-rays) or the emissions of radioactivity can cause the release of energy referred to as ionization, which generates "free radicals" (i.e., electrically-charged molecules deficient of an electron). These, in turn, can react with DNA or other cellular components to induce mutations or other biological alterations.[26]

Uranium miners are exposed to ionizing radiation in the form of alpha particles. X-ray technicians are exposed to ionizing radiation in the form of gamma emissions. The different mechanisms of action of the two types and their implications with respect to cancer development are discussed in Part B of this chapter. Suffice it to say that both types can cause mutations and produce cancer.

Thus, cancer can be caused by physical agents such as radiation, or by chemicals. (Viruses may play some role, but are of minimal significance). Chemicals can act directly on the genetic material in the cell, or may have to be converted into some other substance in the body which ultimately does the damage. Substances can be mutagenic without necessarily being carcinogenic. In other words, not all mutations result in uncontrolled growth. Also, substances can be carcinogenic without being mutagens.

Exposure to carcinogenic chemicals may occur in the workplace, from medicines, or from the general environment. The relative role played by the various carcinogenic agents is hotly controversial as will be described below.

2.3 *Factors Influencing the Latency Period*

Although it is most important to determine the agents capable of initiating the cancer process, it is also important to appreciate the later events which may be rate-limiting in terms of setting the incidence of a particular tumour.

It is widely known that the time between exposure to a carcinogen and the actual development of the tumour is lengthy and variable. This latency period, as it is called, will obviously be affected by the end-point used to measure it, i.e. diagnosis or death. Both end-points have their limitations, as there are a number of factors that determine the speed with which the diagnosis is made as well as many variables determining the time of death. Also, in the presence of a high background rate of cancer, it is difficult to determine when the increased incidence actually begins to occur.

Even discounting these factors, however, the latency periods associated with various cancers are quite variable. A review of the literature on latency periods conducted for this Inquiry by the Canadian Centre for Occupational Health and Safety (CCOHS)[27] has stated that the latency for bladder cancer ranges from 1 to 52 years with the range of averages being 15 to 19 years; latency for leukemia ranges from 1 to 46 years with averages of 4 to 15 years; lung cancer latency ranges from 4 to 47 years with an average of 13 to 40 years; and nasal sinus cancer has been associated with a longer latency, ranging from 10 to 70 years and averaging 22 to 45 years in various studies. (Possibly cancer cases at the low end of the range are not due to occupational exposures).

The factors influencing latency include: 1) the total dose, 2) the dose rate, 3) the age at first exposure, 4) sex, 5) lifestyle factors such as nutrition, smoking and general health status, and 6) occasionally hereditary factors. The studies cited as best illustrating that latency is a true biological phenomenon are those concerning atomic bomb survivors. According to this review the increased incidence of leukemia was first seen after about 2 years from the time these people received this single large dose of radiation. The incidence of leukemia peaked after about 4-5 years and has since decreased to near background levels. In contrast, an increased incidence of lung cancer first began to appear only after about 15 years and has apparently still not peaked. (Appendix 2 contains a list prepared by the CCOHS of latent periods associated with known human occupational and environmental cancers.[28]) The variability in latency periods poses a difficulty for the compensation system in trying to assess the probability of work-relatedness of a claim. It also poses considerable difficulty for epidemiological studies which consequently require extremely long follow-up of populations exposed to possible carcinogens in order to determine the true risk.

One explanation for the latency period on the cellular level is that carcinogenesis is a multiphasic process. According to this theory the first phase is termed initiation. The "initiated" cells appear not to be tumour cells, and they cannot be recognized directly. A subsequent phase - promotion - causes them to develop into tumour cells that replicate to yield gross tumours. Although promotion is often considered to be a single phase, it requires a prolonged period, i.e. the latent period, and probably comprises more than one process. When a group of normal cells are exposed to a carcinogen, for example benzo(a)pyrene found in coke oven emissions, this does not immediately induce neoplasia. There can be a lag of months or years or even decades, during which changes in cell properties occur (which frequently include hyperplasia, metaplasia and other "pre-malignant" stages) then ultimately the fully malignant tumour. A very low dose of benzo(a)pyrene on mouse skin may not result in a detectable increase in cancer, but when this is followed by repeated applications of a tumour promoter (such as the phorbol ester known as TPA) then a large number of skin tumours are induced.[29]

In testing systems, chemicals that are recognized as carcinogenic must have initiating and promoting activities either directly or through one or more metabolites or by-products, unless one or both of these functions are provided from other sources.

An initiating chemical has the special property of being a strong electrophilic reactant. Evidence strongly suggests that initiation is an induced change in the informational content of the genetic apparatus (DNA) and takes place rapidly and irreversibly. Promotion, on the other hand, does not involve a genetic mutation, occurs over a prolonged period, and is at least partially reversible. It may act by altering growth properties, increasing or decreasing levels of certain enzymes, and modifying membranes of cells, facilitating certain results. (These properties are tabulated in Table IIA-8 along with a schematic representation of this process, Figure IIA-4.)

Table IIA-8

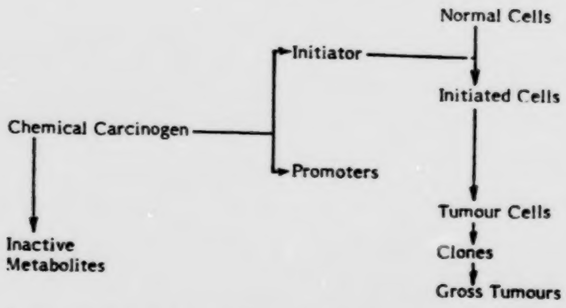
A Comparison of Biological Properties of Initiating Agents and Promoting Agents

Initiating Agents	Promoting Agents
1. Carcinogenic by themselves "solitary carcinogens."	1. Not carcinogenic alone.
2. Must be given before promoting agent.	2. Must be given after the initiating agent.
3. Single exposure is sufficient.	3. Require prolonged exposure.
4. Action is "irreversible" and additive.	4. Action is reversible (at early stage) and not additive.
5. No apparent threshold.	5. Probable threshold.
6. Yield electrophiles--bind covalently to cell macromolecules.	6. No evidence of covalent binding.
7. Mutagenic.	7. Not mutagenic.

Source: Weinstein, I.B., Evaluating Substances for Promotion, Co-factor Effects and Synergy in the Carcinogenic Process. J. Environ Path and Tox 3:89-101, 1980.

Fig. IIA-4

A Simplified Scheme for the Steps in the Induction of Cancer by a Chemical



Source: Miller, E.C. and Miller, J.A., Mechanisms of Chemical Carcinogenesis Cancer 47: 1055-1064, 1981.

Some chemicals have both initiating and promoting activities and are therefore considered to be "complete carcinogens". In these cases the initiating and promoting activities of a given precarcinogen can theoretically either be from the same or from different metabolites of the carcinogen. The necessity for promotion as well as initiation is invoked to explain the long latency period in some tumours.

Thus although it is the initiator that produces the genetic change that ultimately produces the cancer, the presence or absence of promoters will affect the rapidity with which the cancer develops, and hence even whether or not it becomes manifest. Hence all 6 variables mentioned above may affect tumour development and should be taken into account in assessing the plausibility of a given latency period.

2.4 The Multifactorial Nature of Carcinogenesis

With even the minimal understanding that does exist with respect to mechanisms of cancer development, it is clear that many factors are involved in determining if a given carcinogen will ultimately result in tumour formation.[30] The particular characteristics of the carcinogen, the presence of other carcinogens and the cumulative dose are certainly of importance.

The interactions between factors may increase, perhaps multiplicatively, the incidence of cancer associated with one or other of the factors. This phenomenon, whereby two or more agents that can act independently as carcinogens act synergistically, i.e. in more than an additive fashion in the same host tissue, is termed "syncarcinogenesis". One factor may, in fact, be non-carcinogenic in the absence of other factors but may enhance the action of carcinogens when present simultaneously with a known carcinogen. These are called co-carcinogens. (These differ slightly from promoters which exert their effect after carcinogenic initiation has occurred.)

Immunological influences and genetic capability of repairing DNA mutations should not be discounted completely, as there is some evidence of their importance, at least in individuals suffering immune deficiency syndromes or taking immunosuppressant drugs. There is also evidence suggesting that dietary factors play a role in the process of carcinogenesis, e.g., evidence suggests that Vitamin A deficiency renders some epithelial tissues more prone to carcinogenic effects. It is now, however, generally accepted that although genetically determined differences in susceptibility do exist, the extreme hypothesis that there is a small proportion of the population who are highly susceptible while the rest are not susceptible at all, is unfounded.

Thus "co-factors" include nutritional aspects, metabolic, immunological and occasionally genetic factors. As it is now undisputed that most cancers have environmental causes, it is thus the range of environmental carcinogens, acting together and influenced by numerous co-factors as well as promoters that determine the ultimate result. The argument that many individuals exposed to occupational carcinogens have life style habits contributing to their cancer has been inappropriately used to under-rate the importance of occupational exposures. However, recognition that many exposures of different lengths and intensities can contribute to any cancer, emphasizes how difficult it is to attribute a given case of cancer to any single cause.

2.5 *The Threshold Debate*

It is also undisputed that carcinogenicity is dose-dependent - i.e., the greater the exposure to a carcinogen, the greater the likelihood that a tumour will result. With the increasing understanding of the derivation of tumours from single cells acted on by mutagenic carcinogens, Peto has pointed out that the decade-long argument that "threshold dose levels of carcinogens must exist below which the general population is absolutely safe" is finally being abandoned.[31] The Delaney Amendment to the U.S. Food and Drug Act, for example, prohibits the deliberate addition to foods of chemicals that have been shown to be carcinogenic in man or animals; the Federal Insecticide, Fungicide and Rodenticide Act prohibits use of pesticides that have been shown to be carcinogenic, and apparently, proposed OSHA rules would have prohibited industrial use of carcinogens unless alternatives were unavailable, and then would have required exceptionally strict control of exposures.

Nonetheless, some proponents of the threshold hypothesis argue that limited quantities of many carcinogens may be virtually safe, and therefore from the compensation point of view, in cancer victims who had been exposed to only a small amount of carcinogen, the cancer could not possibly be attributed to the exposure. (Figure IIA-5 depicts several possible relationships between cancer incidence and concentration of the carcinogen.)

One proponent of the threshold theory, Peter Gehring of Dow Chemical Company, has inferred that because 95% of chemicals have the capability of interacting with DNA, the fact that not everyone gets cancer indicates that the body must have a sophisticated system of dealing with potential carcinogens. He points out that contributors to the body's defence system include membranes to restrict carcinogens from contacting the DNA, detoxification systems, enzymes for DNA repair and an immunological system that destroys aberrant cells. He also notes that there are naturally-occurring antagonists for cancer, particularly antioxidants such as Vitamins A and E.[32] (He fails to note, however, that although a great many chemicals have the capability of interacting with DNA, only a very small number are capable of inducing mutations or irreversible damage that has bearing on the replication process.[33])

Another proponent of the threshold theory, Paul Kotin[34] of Johns-Manville uses estrone as an example. Suspected of being carcinogenic in humans in large amounts, it is nevertheless present in very small concentrations in all humans. Schneiderman of the National Cancer Institute (NCI), however, has pointed out that estrone is present in much higher concentrations in women than in men and that this might be at least part of the cause of the much greater incidence of breast cancer in women.[35] Kotin also argues that the presence of known carcinogens such as nickel and chromium in trace concentrations in all humans is further evidence that small quantities of carcinogens do no harm and that it is only when the protective system is overwhelmed that the chemical presents a threat. To this Wilson has pointed out that a substance can be necessary for one part of a body's function while providing the risk of destruction to another.[36] Others assert that even if the active carcinogen is not detoxified, a large proportion of it can react with DNA without initiating tumour formation and that the probability of a carcinogen reacting with exactly the right site must be quite low for very small doses. Cornfield[37] presents an elaborate mathematical model based on the existence of a detoxification system, to illustrate the threshold hypothesis.

The last piece of evidence that is frequently cited in support of the threshold hypothesis is the relation between the dose of a carcinogen and the latent period. As it is generally accepted that for many carcinogens the latent period increases as the dose is reduced, some argue that at very low doses the latent period is longer than the animal's lifetime, so it is essentially a threshold level. The fact that there have been experiments in which very low levels of known carcinogens failed to produce cancer in experimental animals has been cited by Mastromatteo as one of the reasons he believes in thresholds.[38]

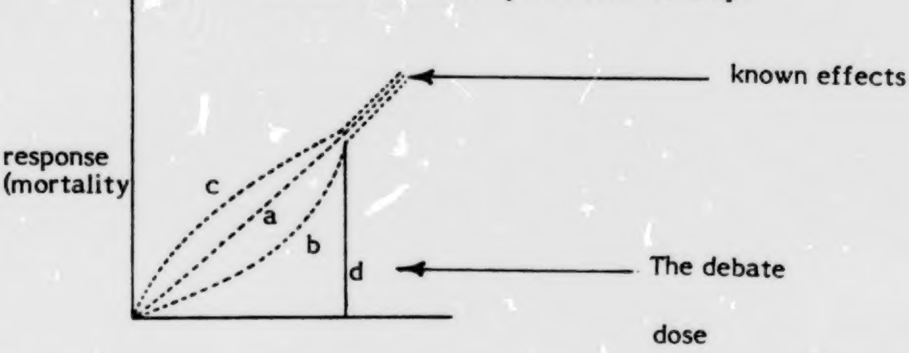
On the other hand, the notion of thresholds has been severely criticized by acknowledged experts, including Sir Richard Doll and Richard Peto of Oxford University and Schneiderman of NCI. They point out that the relationship illustrated by Cornfield is a mathematical artifact, resulting from the fact that the incidence of tumours in animal studies is very high, often approaching 100%.[39] Peto has shown that if there is already so much of a pollutant that cancers produced by it are common, an additional dose of this pollutant or any dose of a pollutant which gives cancer by the same mechanism produces a linear effect in the incremental dose. (This is shown graphically in Figure IIA-6.) He states that since the cancer rate is so high anyway, it is likely that for many pollutants the concentrations are already above a threshold (if any) and using a linear dose response curve for low dose increments of "new" carcinogens does not appreciably overstate the effect.[40]

The no-threshold theory originated from the understanding that cancer is an expression of a permanent, replicable change in cellular genetics resulting from the interacting of one molecule of carcinogen with a critical receptor in one cell. According to the "single hit" or linear hypothesis, at low doses the dose-response curve for chemical carcinogens is a straight line that would go through zero dose, and, if there were no spontaneous incidence of tumours, zero response. More sophisticated modelling systems also adhering to the no-threshold theory, assume that two or more events are necessary for induction of a tumour, and therefore predict a dose-response curve that is concave upward, with a slightly lower incidence of tumour predicted at low doses as compared to the single hit theory.

Applying the multistage theory of carcinogenesis, and taking into account variable carcinogenic potencies, Doll developed a formula to describe the relationship between dose and cancer incidence.[41] It takes into account the fact that there is a latent period in cancer development, it can predict synergism whereby cancer incidence can be proportional to the product of the concentration of two substances instead of their sum (as is the case for asbestos and smoking, for example) and it can take into account co-factors. It is based on the fact that the probability of each insult is proportional to the concentration of the carcinogenic substance,

Fig. IIA-5

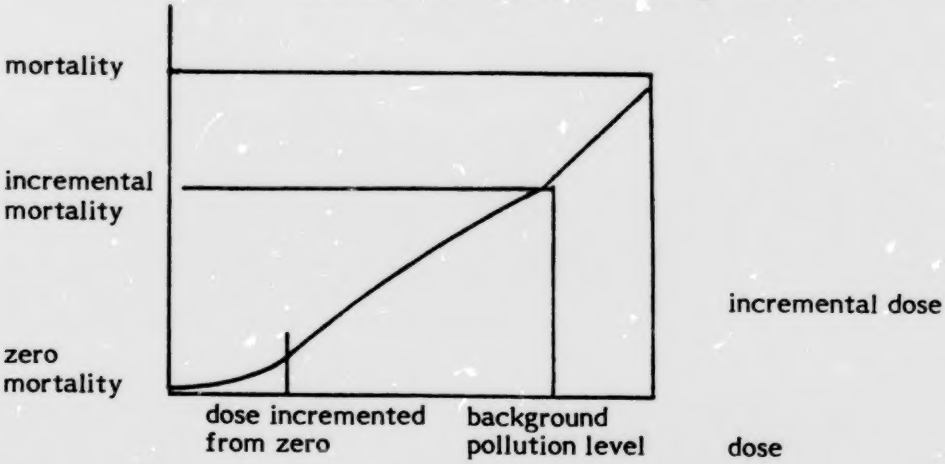
Diagrammatic Representation of the Various Theories on Dose-Response Relationships


known effects
response (mortality
c
a
b
d
The debate
dose

a 'linear theory'
b variation
c low dose proportionately more dangerous
no threshold
d threshold theory

Fig. IIA-6

Possible Effect of a Small Dose of Carcinogen on Mortality in the Presence and in the Absence of a Background Level


mortality
incremental mortality
incremental dose
zero mortality
dose incremented from zero
background pollution level
dose

Source: Peto, R. NIEHS Extrapolation Conferences, Pinehurst, 1976.

and to the duration of exposure, thereby showing that the cancer incidence is linear with each individual substance causing an insult. Most scientists have found this "no-threshold" formula to be consistent with present knowledge. Evidence in support of the no-threshold theory includes the exhaustive data showing a linear dose-response curve for radiation-induced cancer and the evidence on cigarette smoking, as well as evidence from animal experimentation.

The data on radiation carcinogenesis come from a variety of sources, including atom bomb survivors,[42] workers who painted luminous dials with radium,[43] patients who received radiation treatment for ankylosing spondylitis[44] and children whose mothers were given diagnostic x-rays in pregnancy.[45] Although some animal studies suggest a low dose response slightly less than linear[46], all the data are consistent with the absence of a threshold. (This is reviewed in depth in Part B of this chapter with respect to the role it plays in the adjudication of cancer claims from radiation-exposed workers).

A linear response has also been demonstrated in cigarette smoking. A study of British physicians[47] showed that 40 cigarettes a day gave 10 times the incidence of cancer of 4 cigarettes a day. (Admittedly there is no data at very low levels such as 1 per year). Many animal studies are consistent with the absence of a threshold, including the "Megamouse" study.[48] A complex chronic study utilizing 24,192 mice which were fed the carcinogen 2-acetylominifluorene (2-AAF) was conducted by the National Centre for Toxicological Research to study dose-response relationships at very low levels. The incidence of bladder tumours was found to drop off sharply as the dose was reduced, however as time was extended, bladder tumours did indeed appear with no indication of a threshold. Moreover, liver tumours showed a linear response over the experimental dose range, thereby very clearly dispelling any notion of a threshold.

It is pointed out, though, that a statistical analysis of standard animal carcinogenicity experiments does not now, nor will it probably ever resolve the threshold question. This debate, although for the most part abandoned, is perpetuated by problems inherent in assessing carcinogenicity and will be discussed below. In the absence of scientific certainty, the prudent approach is to accept the no-threshold model and assume no safe limit. Suffice it to say that the more important point in the real world of multiple exposures, is that each additional exposure to carcinogens, no matter how small, will contribute to the total carcinogenic effect. This latter point is beyond dispute and certainly detracts from the importance of the threshold debate.

More importantly, despite the fact that the threshold question often emerges in compensation battles, it really offers little guidance as to the work-relatedness of any particular cancer claim. Even if there was unanimity that any exposure to a carcinogen could result in cancer, or at least increase the probability of developing cancer, it still would not imply that all cancer cases that occur in the presence of that exposure are "attributable" to it. Thus the compensation system must arbitrarily decide what increase in probability of developing cancer it will require before deeming the occupational exposure to be responsible for all cancers that occur at that level. The controversy surrounding this issue will be discussed in Part B of this chapter.

2.6 *Multiple Causes - Single Disease*

The last point that must be mentioned in order to appreciate the complexity of the issue of cancer causation, is straightforward and by this time, fairly obvious: there is very little to distinguish tumours resulting from different carcinogenic agents. Except in a few unusual situations, such as the strong association between mesothelioma and asbestos, or liver angiosarcoma and vinyl chloride, the pathology caused by workplace exposure to carcinogens is not particular to that etiological agent. Certain factors, such as a tendency of occupationally induced tumours to develop at an earlier age than tumours which arise "spontaneously", or a noted continuous or repeated exposure to specific substances, may alert a physician to a possible occupational connection in an individual patient.[49] However, the characteristics of current medical practice and all the diagnostic, etiological and research issues discussed elsewhere in this report are certainly major factors in contributing to and perpetuating the uncertainties in cancer causation, and make any firm distinctions in individual cases very tenuous.

3. Difficulties in Assessing Carcinogenicity

3.1 *Limitations of Epidemiology*

Human carcinogens, by definition, can only be identified with certainty by epidemiological studies in man. This procedure is manifestly too late to prevent suffering and therefore is considered by many to be unsuitable as a method on which to base regulatory measures. Moreover, the epidemiology of cancer is exceedingly complex. It involves the search for clues to differentiate cancer patients from those who have not contracted the disease. Due to the unique nature of cancer, with its long latent period and undifferentiated growth of cells, these clues are often scarce and difficult to interpret. Cancer epidemiology is hence fraught with uncertainty and bitter controversy.[50]

Evidence of carcinogenicity from human studies comes from three main sources:[51]

1. Case reports of cancer victims who had previously been exposed to a specific chemical or process.

2. Descriptive epidemiological studies in which the incidence of cancer in human populations is found to vary spatially or temporally with exposure to the agents.

3. Analytical epidemiological studies (cohort and case-control) in which exposure to the agent is found to be associated with an increased cancer risk.

The first category may alert the community to the existence of a possible problem, but is not considered to be of sufficient rigour to allow assumptions about causation. Descriptive studies would include the much publicized cancer maps. The fact that there are significant geographical differences in cancer rates has added considerably to knowledge in the area of cancer causation, especially with respect to the role of the environment. Although the differences themselves do not tell us whether environmental or genetic factors were involved, the fact that studies have shown that when people migrate from one area to another, their disease pattern tends to adjust to that of the country to which they migrate, has provided strong evidence for the environmental rather than genetic, causation of cancer. Similarly, correlations have been made between cultural or socioeconomic variations and specific cancer rates, to yield certain inferences regarding causation. At best, though, these data can be used to obtain clues as to possible high risk groups, which must be further investigated by other methods.

The more sophisticated epidemiological methods, however, have their own complexities and difficulties. Some of the more common of these can be summarized as follows:

a) Requirements for large study population: Epidemiological research requires the assembly of a large enough number of cancer cases of a particular type from which to draw valid conclusions. Although lung cancer, for example, is a major killer, the probability of any individual contracting lung cancer in any given year is still relatively small and therefore a large number of people would have to be observed to ensure enough cases of the disease to study to satisfy the statistical requirements. Identifying a sufficient number of cancer cases together with their medical and exposure histories is often quite costly and difficult. Cancer registries, where they exist, help in this endeavour. (The availability of data in Ontario is discussed in the next section.)

   Several epidemiological techniques are available. The most sophisticated, the randomized controlled trial, cannot be used in toxic exposure studies as it is obviously unethical to subject an individual to an exposure in order to study the result. The follow-up or cohort study, which consists of following a large number of healthy people for several years in order to determine which of them contract and die from a specific disease, is also a powerful method. The number of deaths observed among people in a specific category is divided by the number expected to die in that category, yielding a value known as the Standardized Mortality Ratio (SMR). For occupational cancer, the variation of this technique which is used is called the historical or retrospective follow-up study. In this method, the cohort of workers is defined to be all those engaged in a particular process at some time in the past. In the follow-up a variety of documents must be searched, such as employment, social security or motor vehicle records to ascertain the vital status of each cohort member at some later time. Causes of all deaths are obtained from death certificates, (consequently inaccuracies in diagnosis may bias the results). Then, based upon actuarial tables, the SMR is calculated. The greater the SMR, the stronger the inference of carcinogenicity. If the cohort size is too small, then even a greatly increased risk of cancer cannot be detected statistically.

   Another form of epidemiological study, the case-control study, depends on locating patients with a particular disease (the cases) and another group of people without the disease and asking them the same set of questions. A figure called Relative Risk is estimated from the replies. This approach is limited by the fact that it relies on knowledge of exposure from many years earlier. As will be discussed below, this presents great difficulties.

b) Establishing an appropriate control group: In any comparative study, the control or comparison population must be appropriately chosen. For example, the finding of a low mortality rate in a group of workers as compared to people of similar age in the general population, does not mean that the study group is free of excess risk. The general population includes disabled and chronically ill individuals while the industrial population in question is at least healthy enough to have been hired. This phenomenon is known as the Healthy Worker Effect. It is thus suggested that a better control population is a group of workers from other industries or the same industry where there is no exposure to the suspected carcinogen. Problems may arise because other potential control groups may themselves be exposed to carcinogens. The fact that the general population, (the control used in most occupational cohort mortality studies) includes many individuals exposed to some of the same carcinogens found in industry, results in an understating of the risk of exposure. (This would be analagous to comparing the risk of lung cancer in smokers to that of the general population, which includes a large percentage of smokers, instead of comparing the risk of smokers with that of non-smokers.)

c) Requirement for adequate follow-up The long latency period, which for some carcinogens is decades, poses difficulties in yet another manner. Failure to follow the cohort for a sufficient length of time can result in serious errors in computing cancer death rates. If a greater proportion of living retirees is found as compared to possibly dead ones, underestimation of the death rate results. Furthermore, if the cohort is followed for too short a period of time, i.e., with inadequate time allowed for the latency period to lapse, a deceptively reduced cancer mortality rate will also result. (This was the case for minimizing the link between benzene and leukemia based on an average follow-up of a mere three years.[52])

d) Difficulties in obtaining adequate exposure data: As alluded to above, perhaps the most serious limitation of epidemiology, and one that plagues even the best designed study, is the inability to obtain adequate data on exposure to carcinogens at times which preceded the finding of cancer by many years. In fact it is difficult to obtain current exposure information (as will be discussed in a later section). Unless specific measurements were recorded, which is rarely the case, it is impossible to reconstruct the extent and even the content of industrial exposures, as often the original process has radically changed or methods of control have since been instituted.

e) Difficulties in controlling for confounders: Another source of controversy is sorting out whether an association really reflects a causal relationship or whether the association is due to confounding or mere chance. Tests of statistical significance address only the question of chance. Recognizing the key or causative factor from amongst irrelevant or merely associated ones is more difficult. Although methods have been devised and used successfully to measure confounding, it is impossible to control for unknown or unsuspected risk factors. There are numerous examples of associations between certain habits, characteristics or exposures with a particular disease which have wrongly been considered to be the causative factors for that disease. The most commonly recognized variable to control in assessing the effects of occupational exposures is cigarette smoking. Unfortunately, many studies of occupational exposures have failed to control for smoking history, and it has been pointed out clearly that many studies on the effect of smoking have failed to control for occupational exposures.[53]

Despite these limitations, there are a number of criteria which can be applied to the results of a study or series of studies to assess the link between exposure and cancer. These criteria for causality, as they are called,[54] include: 1) the strength of association, that is, the relative risk and degree of statistical significance; 2) the consistency or extent to which similar effects have been seen in groups with similar exposures; 3) the temporality, that is, whether or not the relationship between the time of exposure and development of the disease is reasonable in terms of what is known regarding the latency; 4) the dose-response relationship, or the relationship between intensity of exposure, frequency of cancer, and latency; 5) plausibility, that is, the existence of a reasonable hypothesis to account for the finding; 6) coherence of the association, that it does not conflict with what is already known; 7) specificity, that is (to the degree that it is possible) the association of the factor with one disease; and 8) experimental evidence, or the extent to which animal and cellular studies have shown the association. This last point is discussed further below.

3.2 Animal Studies and Short-term Tests

Due to the expense and difficulties involved in epidemiological studies, attention has been concentrated on developing laboratory methods to identify chemicals by their biological activity as likely human carcinogens. Moreover, this has the distinct advantage of being able to determine carcinogenicity without depending upon the results of human exposure.

These lab models include both "short-term" in vitro (or test tube) tests and the more conventional chronic animal studies. Taken in conjunction with suggestive epidemiological indicators they can be powerful tools, however it is unlikely that these tests could ever do away completely with the need for epidemiological studies. Although a great deal of knowledge about cancer has been derived from animal models, not surprisingly, the use of this knowledge has been the source of considerable controversy.

The battery of short-term tests include the well-known Ames bacterial mutagenic assay, with possible sophistications such as the fortification of the culture medium with actual tissue preparation or even activation by in vivo (or live) animal metabolism. Other techniques include the transformation of mammalian cells in culture, also possibly with tissue extract activation, and other indicators of DNA damage such as karyotyping, DNA repair assays, or sister chromatid exchange. The predictivity and reliability of these tests are highly dependent on the structural class of chemicals being tested. Also consideration must be given to the choices that experimentalists must make in selection of the protocol, wherein minor modificatons may produce different outcomes.[55] The numerous false positives and false negatives limit the usefulness of these tests and certainly a negative result cannot exclude the possibility of carcinogenicity. In spite of advances in short-term assays and their undeniably important role in screening some categories of chemicals, the ability of chemicals to induce cancer in the whole animal, more closely approaches carcinogenic potential in humans.

Most scientists now argue that if a chemical causes cancer in well-designed animal tests there is a strong likelihood that it will also cause cancer in exposed humans.[56] There is growing evidence that this is indeed the case. The two major criticisms about the use of evidence from animal studies revolve around the recognized difficulties associated with extrapolation from species to species, and from high doses in small experimental populations to lower doses in very large populations. Much of our knowledge about the relative potency of carcinogens is indeed based on animal studies.

a) Species-to-species extrapolation The assumption that what is found to cause cancer in one species must also be considered to potentially cause cancer in others has been fundamental to cancer research, and is based on the fact that life processes, including the basic genetic processes in mammalian and other animals are fundamentally the same as those in humans. Cancer is found in insects, fish and even plants, all of which possess the same cellular ultrastructure, with genetic structure and coding mechanisms. Carcinogenic chemicals thus tend to be similarly active in many different test species. However, there are often differences in the susceptibility of the various species, in different strains within species, and in different individuals in any particular strain. These differences relate to factors such as different enzymatic and detoxification systems. Also, the target organ affected by the carcinogen may vary from species to species.

   Criticism has been levelled against the use of mice, for example, because of their unusual sensitivity and the high incidence of spontaneous tumours among them. These problems, however, can be overcome by proper selection and breeding of strains.[57]

b) Extrapolating from high-dose animal studies to low-dose human exposure: There are some people who assert that the dosages used in animal studies make them invalid to human situations. The animal experimentalists point out, however, that a well-designed test uses a range of doses and results are based on mg/kg of the animal's body weight. If the dose is too high animals risk dying from toxic effects before the cancer has a chance to develop. The defense for large doses is that:

   1) some carcinogens are much less potent than others and that for any given chemical, humans may be more or less sensitive than the test animal to the carcinogenic agent;

   2) finite animal resources restrict the use of large populations of animals.

   Given that a human-level dosage might not produce a detectable result in a small rodent population, even for a carcinogen which may be the cause of many thousands of cases in humans, the only alternative is to increase the dose and thereby increase the probability of inducing cancer in a particular animal.

It must be stressed at this point that animal experimentalists believe that there is no way of predicting human thresholds from animal data. In assessing the potency of a carcinogen, it is also invalid to assume that in all cases potency in humans parallels potency in rodents, although this has been the case for some substances.

In summary, although much uncertainty still exists with respect to defining carcinogenicity, many techniques have been developed and much knowledge has indeed been accumulated. Epidemiology can be a useful tool but it is time-consuming, costly and requires a long lag. The inherent methodological issues which may bias the results must also be recognized.

Animal and short-term laboratory studies have an important role to play despite the controversy with respect to their applicability to human exposures.

Unfortunately, enough scientific basis seems to exist for each side to selectively portray the facts and prove its own argument with an air of scientific confidence. In reality, the degree of caution, or the amount of "proof" required is subject to one's own concerns and biases. Nonetheless, guidelines as to what constitutes adequate proof of cancer risk to humans have begun to be developed which seem to be acceptable at least as a starting point.

3.3 *Internationally Recognized Human Carcinogens*

In 1971 the International Agency for Research on Cancer (IARC) began a program to prepare monographs on carcinogenic risk to humans of various chemicals and industrial processes. World experts in chemical carcinogenesis, epidemiology and related fields gathered together to critically assess the data. Selection of chemicals for evaluation by the IARC was based on two criteria: first, that there were data related to carcinogenicity in humans or experimental animals; and secondly, that there was evidence of human exposure.[58] The vast number of chemicals and industrial processes which have never been studied were obviously excluded from evaluation.

In October 1977 the Working Group met to standardize the evaluation of carcinogenicity studies both in humans and in animals, and made the important recommendation that "in the absence of adequate data in humans, it is reasonable for practical purposes, to regard chemicals for which there is sufficient evidence of carcinogenicity in animals, as if they presented a carcinogenic risk for humans."

Of the 442 chemicals and industrial processes evaluated by the IARC by the end of 1978 it was concluded that there was sufficient evidence of carcinogenicity for 142 (32%) of them. Human evidence, however, was available for only sixty-two of the 142 (14%). Six of these were dropped due to time limitations, leaving only 54 chemicals and industrial processes with data on carcinogenicity from human as well as animal studies to be reviewed by the Working Group which met in January 1979.

The method used in evaluating these 54 substances consisted first of separate assessments of the human and animal evidence which were debated and adopted by the Working Group. The evidence from animal studies was classified as sufficient, limited, inadequate, negative or no data, according to specific guidelines (outlined in Appendix 3). Sufficient evidence of carcinogenicity would entail, for example, experiments in multiple species or strains, multiple experiments (preferably with different routes of administration and varying dosages) as well as perhaps an unusually high incidence, early age of onset, dose-response effect or additional information on mutagenicity of chemical structure. Sufficient evidence of carcinogenicity in human studies indicates a causal association between exposure and human cancer. This in turn implies that no specific bias was identified to explain the association, the possibility of confounding had been considered and ruled out, and the association was unlikely to be due to chance. Although a single well-designed study may suffice, confidence in inferring causality would be increased when several independent studies show the association, when the association is strong, where there is a dose-response relationship or when reduction of exposure is followed by reduction in tumour incidence.

Secondly, an overall evaluation of carcinogenicity for humans was made based on the combined evidence from human and animal studies and the substance classified into groups as depicted in Table IIA-9. Of the 54 chemicals, groups of chemicals, and industrial processes evaluated, 18 were found to be definite human carcinogens from evidence directly from human studies. A further 18 were thought to be probable human carcinogens, although the evidence was considered to be inadequate to conclude an undisputed causal association. This group was further subdivided; for 6 there was a high degree of evidence, and for 12 there was a lower degree. Data on the remaining 18 chemicals were considered insufficient to allow evaluation of carcinogenicity.

Although the IARC monographs are highly regarded in the scientific community, IARC is not the only organization active in assessing the evidence of carcinogenicity. There are 21,000 chemicals listed in the 1976 publication of Registry of Toxic Effects of Chemical Substances by the National Insitute for Occupational Safety and Health (NIOSH) in the U.S. Of these, a subfile was developed of suspected carcinogens which contained 2,400 substances. (Occupational exposure to 16 has so far been regulated by the U.S. Occupational Safety and Health Administration (OSHA).)

Although these assessments have been put to some use in the regulation of carcinogens in some jurisdictions, they seem to exert little influence with respect to compensation, as will be illustrated in Part B of this chapter. The rationale for this reluctance seems to relate to the specifics of exposure and will also be explained below. Certainly the work of such agencies as the IARC can be much more effectively used in policy formulation not only in regulation of carcinogens, but also in compensation decisions.

4. Exposure Estimates and Other Data Sources

4.1 *Estimates of the Extent of Exposure - The NIOSH Attempt*

As explained above, there are hundreds or perhaps thousands of agents suspected of being capable of inducing cancer. Although documentation that exposure has occurred implies an increase in the risk of developing cancer, it alone carries no implication as to the extent of the increased risk or the probability that cancer will indeed result in the individuals exposed. Nonetheless, estimates of the extent of exposure to various substances can provide some idea of the possible magnitude of various health and compensation problems that may occur.

When the Occupational Safety and Health Act came into being in the U.S. in 1970, the National Institute for Occupational Safety and Health (NIOSH), the main government research agency for occupational health and safety, quickly realized that reliable statistics describing health hazards in industry were virtually non-existent. One project initiated by NIOSH, the National Surveillance Network (NSN) was found to have several shortcomings. Based on state industrial hygiene inspections the NSN lacked consistent reporting, relied on inspected plants only and lacked a statistical basis. To address these deficiencies NIOSH instituted a National Occupational Hazard Survey (NOHS) aimed at recording specific potential hazards, so as to assess what occupational groups were exposed to what hazards, where these occurred, and what the substances were to which the greatest number of people were exposed.

Between 1972 and 1974, visits were made to approximately 5,000 workplaces, selected to provide a representative cross-section of industry type and size. The team, consisting of 20 engineers, collected general information about each plant, such as its major product or service, the number of employees, and the availability and type of monitoring. The most crucial and informative part of the survey consisted of a walk-through inspection in which the engineer or hazard surveyor recorded specific exposures according to precise guidelines set out in the NOHS manual.[59]

A number of problems emerged. Difficulties were encountered in recording the number of workers exposed to specific substances because many products were known by trade name rather than by chemical composition. It was found that companies surveyed were often not aware of the chemical composition of the substances used in their plants. In fact, over 70% of the exposures identified were recorded as trade name products for which the chemical composition was not known to the company. More than 10,000 manufacturers were contacted to identify precisely what chemicals were contained in these trade name products. However, it was found that manufacturers designated as trade secrets more than one third (35.4%) of the products containing a cancer-causing agent. By the date of the congressional hearing on industrial exposure to carcinogens, chemical composition was determined for only slightly more than half of those trade name products.[60]

Three assumptions therefore had to be made in estimating the number of exposed workers:

Table IIA-9

IARC Grouping of Carcinogens and Probable Carcinogens (1980)

The Working Group concluded that the following 18 chemicals, groups of chemicals, and industrial processes are **carcinogenic for humans** (Group 1):

4-Aminobiphenyl
Arsenic and certain arsenic compounds
Asbestos

Manufacture of auramine[1]
Benzene
Benzidine
N, N-bis (2-chloroethyl)-2-naphthylamine (chlornaphazine)
Bis (chloromethyl) ether and technical grade chloromethyl methyl ether
Chromium and certain chromium compounds[1]

Diethylstilboestrol
Underground haematite mining[1]
Manufacture of isopropyl alcohol by the strong acid process[1]
Melphalan
Mustard gas
2-Naphthylamine
Nickel refining[1]

Soots, tars and mineral oils[1]

Vinyl chloride

The following 18 chemicals and groups of chemicals are **probably carcinogenic for humans** (Group 2):

Group A (six chemicals)
Aflatoxins
Cadmium and certain cadmium compounds[1]
Chlorambucil

Cyclophosphamide
Nickel and certain nickel compounds[1]
Tris (1-aziridinyl) phosphine sulphide (thiotepa)

Group B (12 chemicals)
Acrylonitrile
Amitrole (aminotriazole)
Auramine
Beryllium and certain beryllium compounds[1]
Carbon tetrachloride
Dimethylcarbamoyl chloride

Dimethylsulphate
Ethylene oxide
Iron dextram
Oxymetholone

Phenacetin
Polychlorinated biphenyls

The following 18 chemicals and groups of chemicals **could not be classified as to their carcinogenicity for humans** (Group 3):

Chloramphenicol
Chlordane/heptachlor
Chloroprene
Dichlorodiphenyltrichloroethane (DDT)
Dieldrin
Epichlorohydrin
Haematite
Hexachlorocyclohexane
(technical grade HCH/lindane)
Isoniazid

Isopropyl oils
Lead and certain lead compounds[1]
Phenobarbitone
N-Phenyl-2-naphthylamine
Phenytoin
Resperpine
Styrene
Trichloroethylene

Tria(aziridinyl)-**para**-benzoquinone (triaziquone)

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[1] The specific compound(s) which may be responsible for a carcinogenic effect in humans cannot be specified precisely.

1. That toxic substances exist in the remaining 50% of trade name exposures in approximately the same percentages as in those which had been translated into their chemical composition.

2. That the proportion of potential exposure among non-trade name substances is approximately identical to that among the trade name substances.

3. That trade name substances not analyzed for content to date and non-trade name exposures affect a completely different group of workers than trade name substances already analyzed for content.

The authors acknowledged that there are certain inherent weaknesses in these assumptions. The dependence of the estimates on trade name data, necessitated by the time constraints of the study, would underestimate exposure to basic material such as asbestos, unlikely to be hidden by trade names. They believed, in fact, that the effect of the third assumption which would tend to overestimate the number of exposed workers, would not be sufficient to offset the underestimation caused by reliance on trade name data. (For example: whereas some estimates are that as many as 1 million workers are exposed to asbestos, only a total of 880,000 workers were estimated in the NOHS).

Apart from the problems created by the Trade Secrets Act and the widespread use of trade name products, the lack of a uniform labelling system made it difficult for workers to know the substances they were exposed to. Also, in the NOHS no attempt could be made to determine the actual exposure of individual workers. Rather, potential exposure was determined by the presence of substances at the worksite. If, however, the substance was present only in a closed system from which no exposure could occur, no potential exposure was recorded. Potential exposures were divided into two categories: 1) Part-time, defined as a potential exposure of at least 30 minutes/week and not more than 4 hours per day; or 2) Full time, defined as a potential exposure of 4 hours or more per day. (One criticism of the NOHS is that this failure to more specifically take into account duration of exposure, and the lack of measurement of level of exposure, minimizes the usefulness of this effort.)

Based on the survey, it was estimated that more than 7 million U.S. workers were exposed to trade name products containing an OSHA regulated toxic substance and more than 300,000 workers were exposed to trade name products containing one of the (then) 16 regulated carcinogens. As this represented only one half of the trade name products, it was roughly estimated that closer to 600,000 workers were actually exposed.

It was noted that a single worker may be exposed to more than one carcinogen and that interactive effects of certain mixtures can be more toxic than any of their separate components. For example, rust preservatives (nitrites) and emulsifiers (amines) used in some cutting and hydraulic fluids, have the potential to combine to form nitrosamines, a potent class of carcinogens.

Of the total 7.5 million people exposed to a regulated substance or carcinogen, approximately 5% were exposed full-time and 95% were exposed part-time. Although the full-time exposure to carcinogens of up to 44,000 is most significant, the authors felt that part-time exposure to carcinogens involved repeated peak exposures and thus should be considered equally dangerous.

Their estimates thus consisted of the following:

Number of persons exposed full-time or part-time to OSHA regulated carcinogens	=	Number of persons estimated from NOHS to be exposed to a trade name substance discovered to contain an OSHA-regulated carcinogen	X	$\frac{\text{Total number of trade name substances in NOHS}}{\text{Number analyzed for content to date}}$	X	$\frac{\text{Total exposures in NOHS}}{\text{Trade name exposures in NOHS*}}$
879,839	=	307,606	X	2	X	1.43

(* only 20% of exposures arise from trade name products)

Since the occurrence of toxic effects depended on the intensity, duration and frequency of an exposure, and since simultaneous or repeated exposures to a number of different substances may also have altered toxicity, there was no way to assess what health risks were faced by individual workers.

The authors also point out that the NOHS data were based on a cross-sectional survey of exposures, hence estimates of workers exposed at that time represented only part of the problem. A total estimate of persons at risk should consider workers who have been exposed at some prior time over their total working life.

Approximately 8-9% of workers change occupations in a given year and numerous studies of occupational mobility indicate that 1) movement is most likely to occur between occupations that are closely related in work requirements, 2) occupational mobility patterns have been stable over time, 3) mobility rates are highest for occupations with limited training requirements and 4) mobility shows a strong negative correlation with age.

The authors point out that neither the mobility studies nor the NOHS provide adequate data to determine movement into and out of industries in which exposure to hazardous substances may be prevalent. However they conclude that the number of persons in the general population who have been exposed for some substantial period of time is a minimum 2-3 times greater than for a given point in time represented by NOHS data.

All the obstacles encountered in this survey: the use of trade names, the Trade Secrets Act, the lack of uniform labelling, the absence of precise records of duration of exposure or dose levels, and the mobility of the workers themselves, severely affect not only the survey's ability to estimate the size of the population at risk, but also hinder research linking disease to exposure. Moreover, these same problems are encountered in the adjudication of compensation claims, as will be discussed in Part B of this chapter.

4.2 Occupational Health Data Sources in Ontario

As identified in section 3, the carcinogenicity of a certain substance or process can be determined to some extent through short-term laboratory efforts and animal studies. Although certain measures have been taken to eliminate or at least reduce exposure to known carcinogens, more have come on the market. In order to assess the magnitude of the occupational cancer problem that has resulted from these exposures, epidemiological studies must be relied upon. In turn, the two basic components necessary to perform these studies are 1) data with respect to exposure to the particular agent, and 2) data with respect to outcomes such as death or onset of a disease in those who have been exposed.

Section 4.1 described American efforts to obtain data pertaining to current exposure to carcinogens in the U.S. This section will briefly review the present state of data collection in Ontario, regarding both the assessment of the extent of exposure to carcinogens and the availability of data on occupational diseases which may have resulted or will result from past and present exposures.

With respect to assessing the carcinogenicity of substances, the Premier of Ontario has announced the establishment of a joint centre for toxicology between the University of Guelph and the University of Toronto. He explained that:

> This centre will address the concerns of the public on possible hazards from the manufacture, use and disposal of industrial products. Thus it offers the potential for long-term solutions to problems of industrial chemicals....The centre will also assist the Province's industries in the safe development of new chemicals and products for domestic and export markets.[61]

The Advisory Committee on Occupational Health and Occupational Safety (ACOHOS) recommended that the Ministry of Labour establish a funding target for research of 1% of direct annual costs of accidents and industrial diseases. According to the report of the ACOHOS it seems that the Ministry largely determines its health and safety priorities according to the pressing concerns brought to its attention by government, management and labour organizations. Major emphasis has also been placed upon the research required for the development of regulatory strategies.[62]

Investigation of the extent to which workers are exposed to substances that may be designated under the Occupational Health and Safety Act, has occurred to some extent. However, this has largely been confined to recording the number of workplaces that would be affected by designating the substance concerned, and the size of their work forces. A large-scale project along the lines of the study described above has not been conducted. Some within the Ministry believe that this sort of exercise would be futile as the results would be meaningless without knowledge of the level and duration of exposure and obtaining the latter was felt to be too mammoth an undertaking.[63]

Some sources of exposure data that are presently available in Ontario include the following:[64]

The Occupational Chest Disease Service of the Ministry of Labour: This service currently maintains files on workers exposed to silica, asbestos and isocyanates. (Facts and figures from this source are discussed in the next chapter). The Occupational Chest Disease Service is apparently expanding its recording systems to include workers exposed to other toxic substances.

Data collected in compliance with the Occupational Health & Safety Act: The Act obliges employers to maintain worker exposure records for various biological, chemical and physical agents that are designated under the regulations. So far (1981) only four substances have been designated.

Data from the Workers' Compensation Board: Aside from its records on allowed claims for occupational disease, the WCB maintains a number of indices that are quite useful for epidemiological research. In particular, the Mining Master File which contains the mining histories of 80,000 Ontario miners has been useful in providing information on the relationship between cancer and exposure to radon and its daughter products. (It should be noted that the WCB does not accept responsibility for conducting epidemiological assessments of emerging occupational health problems,[65] as will be addressed in Part B below.)

Data on morbidity and mortality: Data on morbidity and mortality from occupational exposure can be derived to some extent from the Ministry's monitoring programs and from WCB statistics. Further, the Occupational Health and Safety Act obliges an employer to submit a written report to the Ministry of Labour within four days of notification that an employee or former employee has contracted an occupational disease. This information, along with any collected during the medical surveillance of workers by physicians in government and in industry, is being computerized by the Ministry. It is recognized, however, that this effort is of little use in identifying individuals suffering from newly recognized occupational diseases.[66]

The Federal-Provincial Advisory Committee on Environmental and Occupational Health has approached the Canadian Centre for Occupational Health and Safety (CCOHS) with a view to compiling data relating to occupational disease across Canada. As the CCOHS is not able to accomodate the request, an ad hoc committee has been set up to consider to whom notification of the said diseases might best be made.[67]

The identification of mortality and morbidity by occupational group has been described by the ACOHOS as a formidable task, and inaccuracies and omissions in death certificates present major difficulties for collecting reliable mortality data by occupation. Selected occupations and selected workplaces have been the focus of some study, as is discussed in Part B of this chapter, however the choice of these occupations is largely based on concerns identified through the filing of WCB claims.

Data from Statistics Canada and Provincial Registries: Statistics Canada has created two computer files, the Mortality Data Base (MDB) and the National Cancer Incidence Reporting System (NCIRS). The files contain information derived from provincial death certificates, which, in Ontario, are collected and maintained by the Office of the Registrar-General.

Whenever cancer is specified as the cause of death, a card summarizing the contents of the death certificate is sent not only to Statistics Canada, but also to the Ontario Cancer Treatment and Research Foundation. Although the deceased's occupation is set out, this information is usually imprecise. Moreover, it is presently not computerized.

The provincial cancer registries utilizing information from Ontario hospitals are also a potentially valuable source of occupational health data. However, these registries have apparently not been receiving adequate funding in recent years.[68]

Thus, a serious weakness that characterizes the information available in the occupational health field is the lack of high quality mortality and morbidity data that relate to specific occupations and to toxic agents in the workplace. The existing information base also suffers from problems of standardization in the definition, measurement and recording of the data. To alleviate the difficulties engendered by the lack of standardization, it has been suggested that a centralized agency be created to coordinate the collection, maintenance and distribution of occupational health data. The CCOHS has also been approached about undertaking this task.

An issue of considerable importance identified by the ACOHOS is the degree to which researchers should have access to employee work, medical and exposure records. It is argued on one hand that full access is required in order to extract the widest range of data. Opponents of this view contend that unrestricted access to employee records will seriously compromise the privacy of workers, especially if the records fall into the wrong hands. The ACOHOS therefore recommends that only bona fide researchers be given wide access through legislation, with appropriate safeguards in place to ensure that the records are used only for research purposes.[69]

Thus obtaining reliable data with respect to occupational disease is problematic. The lack of exposure records is of particular concern not only for research purposes regarding prevention priorities, but also for policy formulation regarding compensation, and in the adjudication of individual claims. Although these problems have been identified in Ontario, and Canada already has many of the facilities in place to begin to address these needs, a completely satisfactory solution has not yet been found.

5. Estimates of the Fraction of Cancer Related to Workplace Factors

5.1 *Inherent Uncertainties*

The issue of how much of the cancer problem is occupationally-related is one of the most controversial in all of occupational medicine and illustrates quite clearly that analysis of causation has intense political overtones. The range of opinions on occupational cancer is wide. Some assert that occupational factors are linked to 4-10% of all cancer. This view is held by Cole,[70] Wynder and Gori,[71] Higginson and Muir,[72] reiterated by Higginson,[73] and most recently by Doll and Peto.[74] Others such as 10 researchers from the National Cancer Institute (NCI), National Institute for Environmental Health Sciences (NIEHS) and the National Institute for Occupational Safety and Health in the U.S. (NIOSH) have asserted that more than 30% of present or future cancers are attributable to the workplace.[75] Still others, such as Epstein,[76] state that the true figure is much higher, while the medical staff of the Ontario WCB, based on the assumption that occupational cancers recorded at the WCB represent the vast majority of all occupational cancers, state that occupational factors are linked to less than 1% of all cancer.[77]

The fact that the "environment" is at the root of the cancer problem and accounts for probably 90% of all cancer cases is not in dispute.[78] The controversy focuses on the relative contribution of the various components of the environment, or specifically, the relative significance of those factors that are purported to be more readily controlled by the individual and therefore supposedly his or her own responsibility (e.g. smoking, and some aspects of diet) versus those factors relatively less under the individual's control (e.g. pollution, workplace conditions) and therefore a social responsibility. Even this apparent dichotomy is not clear-cut, as it can be argued that social influences play a role in "personal habits", and it is argued by some that the individual is not totally without the choice of where he or she lives or works. The division between the influence of working within a plant versus the effect of living downwind from one presents even greater complexities.

The quest to understand cancer causation, principally to establish and promote prevention priorities, but secondarily also for purposes of compensation, has led to the many "educated guesses" and estimates that serve as the focus of the present debate. Some of the pitfalls inherent in this sort of undertaking are well-known and are based on the scientific reality of multifactorial causation. Other pitfalls stem from the many methodological complexities in cancer epidemiology, which have led some to conclusions such as that depicted by the title of one talk "Epidemiology Applied to Occupational Health Problems: A Cruel Hoax".[79] Still other problems in defining causation are attributable to the lack of systematic data collection, as discussed in the previous section.

Some of the major inherent problems were identified by the U.S. NCI, NIEHS and NIOSH in their controversial Estimates Paper.[80] Most of these concerns have been reiterated in subsequent treatises on the subject.[81] They can be summarized as follows:

a) Limited carcinogenic assessment: Due to the insensitivities of epidemiological surveys and the various difficulties in conducting them, as discussed in section 3, only agents and industrial processes which lead to rather large excess incidences of cancer have been identified to date. A recent study surveying all the epidemiological studies reported over a two year period in two major scientific journals, pointed out that at least a third of these studies would not have been able to detect a cancer risk unless the magnitude of the increased risk was very large.[82] Moreover, of the 368 chemicals known at that time to be carcinogenic in some species and to which occupational exposures were known to occur, the IARC found that any form of epidemiological data existed for only 221.[83] The authors of the Estimates Paper stressed that with so few industries and processes adequately investigated, there is little reason to believe that all major occupational carcinogens have already been identified. Doll and Peto[84] echoed this view:

> The hazards that have been recognized this far tend to be those which increase the relative risk of some particular type(s) of cancer very substantially, and important occupational hazards may quite possibly exist that have not yet been detected because the added risk is small compared to other causes, or because only a few people have been exposed, or simply because a hazard has not been suspected and so not looked for.

Although this conclusion is questioned by some, for example, the American Industrial Health Council (AIHC),[85] organ of the chemical industry, the assertion that few industrial processes have been adequately investigated is undeniable and presents an inherent problem in attempting occupational cancer estimates. Needless to say, it also underlines the importance of stepping up the pre-testing of chemicals prior to their introduction into the workplace.

b) Limitations in data collection: As Doll and Peto have recently re-emphasized, despite the passionate debates that have taken place about the likely magnitude of the number of cancer deaths attributable to occupation, no routine system has yet been adopted for generating reliable information. The problem is further complicated by the fact that since treatment is not dependent on etiology and few physicians have been adequately trained to take occupational histories, occupational factors are rarely sought. The general lack of information concerning occupation on death certificates has been mentioned as problematic, as this would enable at least some screening for gross hazards after the fact.

Suffice it to say that the absence of direct information has led to the situation where any precise estimate of the proportion of cancers that are work-related are but informed guesses. Further inherent problems in arriving at this estimate are listed below.

c) Latent period, age and duration of exposure: It is well known that occupational cancers are characterized by latent periods of up to 50 years. As discussed earlier, this leads to potential underestimates if a cohort is not followed essentially to its extinction. The authors of the Estimates Paper illustrated this point numerically. By assuming a constant multiplicative increase in risk at all ages, they asserted that the number of cancers occurring in an exposed group would increase as the 5th or 6th power of age and therefore the number of attributable cancers occurring by age 50 would be 1/5 or 1/6 of that expected by age 70. They therefore concluded that epidemiological studies, which usually fail to follow a cohort to extinction, enumerate only a small fraction of the total excess cancers attributable to an agent.

Again these conclusions are criticized by the AIHC and others, as is some of the mathematical reasoning presented in the appendix to their paper. The information to confirm or deny this hypothesis with respect to the significance of aging is under debate. Nonetheless, the difficulties posed by the dependence of cancer incidence upon age to at least some extent, and certainly upon duration of exposure, as well as the difficulties posed by the long latency, constitute further inherent problems in carrying out accurate numerical assessments.

d) Changes in exposure patterns: As discussed in the previous section, it is well-known that workers change jobs fairly frequently and that even within the same job, exposure patterns change from time to time. Again, it is extremely difficult to establish accurate exposure histories retrospectively, especially with respect to assessing intensity and duration of exposure in order to accumulate dose-response information. There is no dispute on this point.

(Epidemiological studies are not generally available for groups exposed to low levels of carcinogens. In the absence of evidence to the contrary, it is generally assumed that conditions at the time of study are still applicable. Extrapolating risk estimates from one study population to another is a source of contention as will be illustrated in Part B of this chapter.)

Doll and Peto have also pointed out that given the latency period, it is too soon to be sure whether agents are human carcinogens if they were introduced into industry only during the last 20 years. The graphs developed by Davis and Magee illustrating the phenomenal rapidity of the growth of the industrial production of certain chemicals during the mid-20th century[86] are reproduced in Figures IIA-7 and IIA-8. Doll and Peto conclude, however, that it is too early to tell whether the decrease of certain occupations, discontinuation of the use of certain chemicals and the increase in plant automation and improvements in industrial hygiene practices will counterbalance the effect of introducing many new carcinogens into the workplace. They feel that it is still an open question as to whether the number of occupational cancers around the end of the century will be increasing or decreasing.[87]

e) The fallacy of "one-effect-one-cause" explanations: It was explained in section 2 that cancer is a multi-phasic, multi-causal process. The authors of the Estimates Paper assert that not only are both internal and external factors acting together to produce cancer, but many, if not most cancers are influenced by two or more different external factors. They propose that not only are most lung cancers "attributable to" asbestos probably simultaneously "attributable to" smoking, but that it seems likely that a fraction of cancers which appear to be "attributable to" smoking should also be "attributable to" asbestos, radiation and/or other occupational factors. They therefore are critical of the estimates which classify cancers under single "causes" such that after subtracting 80-90% of lung cancers as smoking-related, one is left with a maximum of 10-20% attributable to all other causes.[88] (Interestingly, critics of the Estimates Paper, many of whom put forward the single causation argument with respect to lifestyle factors accounting for cancer, take issue with the use of "attributable" in this case, and suggest that association, not attribution would be more appropriate. This will be discussed more fully below.)

5.2 *The Different Approaches and Range of Opinions*

Given the great difficulties involved in estimating the magnitude of occupational carcinogenesis, it is not surprising that the estimates that have been made are subject to intense criticism. In 1969 Higginson[89] allocated the majority of cancers to "unknown factors" but acknowledged that known occupational factors accounted for 1% of mouth cancers, 1 - 2% of lung cancers, 10% of bladder cancers and 2% of skin cancers. In 1976[90] Higginson and Muir attributed 1 - 3% of cancers to occupational exposures. Wynder and Gori[91] concluded in 1977 that the general estimate of cancers attributable to occupational exposure ranges up to 10% (see Appendix 4, Table A). In papers published that year Doll[92] concluded that occupational factors were of relatively small importance, but gave no numerical estimate and Cole[93] concluded that a reasonable estimate of occupationally-induced cancer would be less than 15% for men and less than 5% for women.

Although the rationale and methodology used to derive these estimates were never clearly explained, Doll and Peto have suggested that it is likely that all these authors considered each type of cancer separately, attributed a proportion of each to occupation on the basis of a few geographically restricted case-control studies, or, in the absence of any such data, on the basis of clinical impressions, and then combined these figures in proportion to the frequency of occurrence of that type of cancer to arrive at an overall estimate.[94] Davis, Bridbord and Schneiderman have pointed out that many of these estimates relied on "the delphi method" whereby a group of experts polls itself.[95]

In a 1980 symposium on cancer prevention, Higginson again asserted that occupational factors account for no more than 6% of all cancers.[96] He asserted that "where several factors are suspected, it is a matter of judgement which exposure should be regarded as predominant." Higginson attributes the marked differences observed in cancer mortality in the different occupational groups not to workplace exposures but to lifestyle differences.

Recently Doll and Peto, using similar but more elaborately explained methodology, reiterated a 4% estimate for the proportion of cancer attributable to occupation.[97] First they explained their reluctant decision to omit from the term "occupational" such situations as lip and skin cancer due to UV light exposure in outdoor workers, or upper respiratory and digestive tract cancer due to alcohol consumption in bartenders, etc. They then explained their decision regarding the calculation of the proportion of cancers attributable to occupational exposure to ionizing radiation, which "can cause cancer in nearly all organs of the body and that some small effect is produced even by small doses and by doses given at low dose rates." To account for this they stated the necessity of adding some figure (which they calculated to be 58 deaths per year) to the sum of the site-specific occupational estimates of cancer. They then proceeded to classify each type of cancer by site into one of three groups:

1) those types never proved to be due to occupational causes at all,

2) those types where the evidence is weak or inconclusive (for which they decided to allow a token proportion of 1% of male cases and 0.5% of female cases as occupationally-related)

3) those types of cancer which can certainly or almost certainly be produced by occupational hazards under defined conditions.

(These lists are included in Appendix 4, Table B.)

They acknowledged that the evidence was only reliable for bladder cancer. For lung cancer they calculated that 5% was asbestos-related and another 5% was due to occupational exposure to the combustion products of fossil fuels. As they believed that other recognized hazards have relatively small risks or have affected only small groups, the total proportion of lung cancers attributable to occupational factors was set at 15% of male and 5% of female cases of lung cancer. They also believed that the occupational hazards in several of the remaining types of cancer are either small or affect only a minute proportion of the total population. "No firm evidence exists on which exact estimates can be made and we have allocated proportions of between 1 and 10% of cases of the remaining types of cancer on the crude and unreliable basis of our interpretation of the literature and clinical impression."

By proportionally adding up the site-specific estimates they arrive at an overall figure of 4%. In consideration of the data relating to mesothelioma they point out that the absolute minimum proportion of all cancer deaths attributable to occupation is 2 or 3%. With respect to their methodology they explained:

> In developing this argument we have given precise numbers not because we believe that they are individually accurate . . . but so that our numbers may be corrected as further information is obtained from reasonably representative studies of individual types of cancer . . . . Since by far the largest contribution to our estimate is 11,000-odd lung cancers, the chief need is for a nationally representative case-control study of lung cancer in relation to smoking and occupation. Such a study might reveal unsuspected occupational hazards, and until it is done something like twofold uncertainty about the true magnitude of the occupational cancer problem is likely to persist.

(The theoretical and practical details of the sort of study to which they are referring have been recently outlined by Siemiatycki et al.)[98]

The weaknesses of what Doll and Peto refer to as the "stop-gap" methods of estimating the approximate current effects of occupation had been labelled in 1978 by the 10 authors of the NCI/NIEHS/NIOSH Estimates Paper. They pointed out the paucity of supporting details and heavy reliance on "educated guesses", "general estimates" and "personal communications". They also criticized what they refer to as "one-effect-one cause" thinking, which ignores multicausation. Most importantly, they felt that estimates of this nature deal with an inappropriate question by attempting to provide estimates of the fraction of present-day cancer incidence attributable to past occupational exposures. The authors felt that it was much more relevant to re-formulate the question because the most important consequences of exposure in the recent past will not be manifested until some time in the future. They asserted therefore, that the more relevant approach is to estimate the contribution of occupational exposure to known carcinogens in the recent past to present and future cancer incidence.

The Estimates Paper applied what has been termed a population attributable risk percent model (PARP) to estimate the fraction of cancer in the U.S. related to occupational factors. Of the then 26 substances identified as human carcinogens, the Estimates Paper projected possible occupational contributions to cancer for 6 high-volume workplace carcinogens, based on risk factors developed from studies of previously exposed populations. The paper deals at some length with the relationship of exposure to asbestos and the generation of lung cancer, cancer of the larynx and mesothelioma. The authors estimate that over the next three decades roughly 13-18% of the total cancer mortality will be due to work exposure to asbestos. Publications which have dealt with a range of other established occupational carcinogens were reviewed and the report commented that for five of them taken together (arsenic, benzene, chromium, nickel and petroleum fractions), the effect may be comparable with that suggested for asbestos. Thus they concluded that between 23% and 38% of the total cancer mortality in forthcoming decades might be occupationally related - even excluding the effects of radiation and of several known chemical carcinogens.

The authors stressed that although most of the exposure to asbestos had been in the past, most of the predicted effects are expected to be in the future. They pointed out that if the "one-effect-one-cause" approach were used, the occupational origin of most of the asbestos-related lung cancers might well have been attributed to smoking and the asbestos-related cancers of the stomach, esophagus and colon, usually thought not to be of occupational origin, might well have been attributed to other or "unknown" factors. They also noted that the present substantial number of asbestos-related cancers may be overlooked by examination of trends in cancer incidence (or mortality) in the general population for various reasons: 1) two of the major types of asbestos-related cancers, pleural and peritoneal mesothelioma, are usually listed simply as lung cancers or as various abdominal cancers respectively; 2) many asbestos-related cancers are simply attributed to smoking; and 3) any increase in asbestos-related cancers of the stomach and colon would be masked by the other long-term trends in cancer incidence at these sites usually attributed to dietary or unknown factors.

In summary, Doll and Peto's approach was based on estimates by cancer-site. In contrast utilizing existing knowledge the NCI/NIESH/NIOSH estimates were based on estimates of occupational exposure to known carcinogens. Whereas the former approach seriously lacked substantiating studies, the latter suffered from uncertainties with respect to extent of human contact and levels of exposure. Whereas Doll and Peto focused their estimate on current cancers related to past exposures, the Estimates Paper concerned itself with current and future cancers related to recent past exposures. Because their conclusion that 20 - 40% of present or future cancers may be work-related has devastating implications, it has been the focus of much debate.

5.3 *The Debate Surrounding the Estimates Paper*

The controversy over the estimate of the fraction of cancers thought to be related to workplace factors rages on. As discussed above, "educated guesses" based on general knowledge of pathophysiology (i.e. how various agents cause disease) and some demographic factors (eg. age, sex and urban/rural differences), having no regard for the prevalence of actual exposure to carcinogenic agents, resulted in low relative risk ratios. The estimate that "as much as 20% or more" of cancer is related to the workplace, has been criticized from one side as an over-estimate, and from the other as an underestimate.

a) Criticisms of the Estimates Paper as an Over-Estimate: As alluded to earlier, the Estimates Paper prepared by experts from NCI, NIESH, and NIOSH basically looked at the number of workers exposed to the six carcinogens in question, multiplied this by the age standardized cause-specific cancer incidence and their risk ratios, to come up with estimates of the number of excess cancer cases expected to develop. Critics of the Estimates Paper hit at virtually all aspects of the report's methodology.[99] Some of the criticisms included the following: 1) Risk ratios used in the report were taken from studies of workers exposed in 1920's - 1950's, and since conditions are presumably better now, these risk ratios are inapplicable; 2) the estimates of the exposed population were criticized since precise measurements were not made in the NOHS, from which much of the data were taken (see Section 4); 3) the use of incidence rates to estimate mortality was put into question in that all persons diagnosed with cancer do not subsequently die of their cancer (although for the ones in question such as lung cancer, the vast majority do.) To the extent that incidence rates exceed mortality rates, over-estimation would occur. The use of age-standardized rates to predict mortality was also criticized in that the age and sex distribution of the cohorts was not specified; 4) it was stated that the authors "confuse attributable risk with associated risk", objecting to the fact that using the PARP model, the sum of causes of cancer exceeds 100% because if occupational factors are even contributory, the case is included.

Indeed, it was recently shown that the extent of shipyard and other wartime exposure to asbestos seems to have been considerably over-estimated.[100] Although the Estimates Paper utilized the only source of exposure estimates available at the time, NIOSH has now provided more recent provisional estimates of exposure. It indicates that the number of workers exposed full-time to the carcinogens in question is actually only 5.6 per cent of the NOHS estimate of workers exposed full and part-time to these same substances.[101] Thus the incomplete and uneven exposure data have given rise to considerable inaccuracies and have served to discredit the Estimates Paper.

b) Criticisms of the Estimates Paper as an Underestimate: Although the report itself alluded to many reasons which may have resulted in the underestimation of the role of occupational exposure, these reasons were systematically enumerated by Epstein[102] as follows:

> First, the calculations in the report ignore the role of radiation and of some ten epidemiologically recognized

> occupational carcinogens, other than the six considered. Second, the risk ratios considered may be artificially low as they were largely derived from less-than-lifetime epidemiological studies. Third, the report does not consider the many statistical and methodological constraints . . . all of which lead to fragmentation of health and exposure records . . . Fourth, the estimates fail to take account of the many chemicals recognized as carcinogenic in animals . . . Fifth, the estimates exclude high risk occupations with incompletely defined carcinogens, such as steel, rubber and tanning industries. Sixth, the estimates do not adequately reflect conditions in small business where exposure levels are likely to be higher . . . Seventh, the report does not reflect major increases in production of the occupational carcinogens it considered such as benzene, with the likelihood of recently increasing exposure. Eighth, the study examined only a limited number of sites, excluding cancers such as skin and bladder which are known to be occupationally related. Finally, the estimates neglect the possible role of fugitive point-source emissions of industrial carcinogens, as causes for the excess of overall and organ-specific cancers, including lung, bladder, colon, pancreas and breast, in residents of certain highly industrialized countries.

Davis has also recently pointed out that although the Estimates Paper only assessed the effect of 6 carcinogens there are now 18 different industrial processes acknowledged to be carcinogenic. Further, whereas the Estimates Paper had estimated that 12 million workers had been exposed full or part-time to 5 carcinogens assessed, NIOSH has now shown that more than 10 million U.S. workers are indeed exposed to carcinogens, considering only 11 of the 18 listed.[103] (Recent findings attributing various cancers to various exposures are summarized in Appendix 4, Table C.)

Although acknowledging the weakness in some of their data, particularly with respect to their asbestos-related cancer estimate, some of the authors of the Estimates Paper have recently reiterated the validity of the approach and re-emphasized their contention that their estimate is conservative.[104]

5.4 Implications of the Production Pattern of Possible Carcinogens

The AIHC have asserted that if industrial chemicals truly constitute an important cause of cancer, then the current cancer burden should be greater. This view, however, ignores the fact that production and normal use of many carcinogens have reached very high levels only since the early 1960's, and, as shown in Appendix 2, the lag time necessary to see the effects of these increases may well be about 20 years or more. Thus the results of these exposures may now be just beginning to become manifest. As illustrated by Davis and Magee[105] and acknowledged by such scientists as Doll and Peto[106] these findings are worthy of considerable attention.

There has been a tremendous growth in overall chemical industrial activity since the 1940's, with the production of synthetic organic chemicals having doubled every 7 to 8 years. Although annual production statistics are not available for most of the 1,500 carcinogens of the approximately 7,000 chemicals tested, Davis and Magee presented the production history for benzene, perchloroethylene, vinyl chloride and acrylonitrile (see Figures IIA-7 and IIA-8), showing that widespread and high-level human exposure to the substances is likely to have occurred only since the 1960's. With respect to less recent carcinogens such as asbestos and chromate, they argue that these substances are long-lived hence it is the cumulative production that is of concern in any case, rather than annual production. They assert: "In light of the relatively recent increase in the volume of production of some carcinogenic and other hazardous substances that are bioavailable, we conclude that it is not now possible to determine the extent to which chemical exposures will influence future cancer rates."

Hoerger of a well-known chemical company argues that improved industrial hygiene practices to control carcinogenic exposures have been such that the occupational component of total cancer will be significantly less in the year 2000 than it is today.[107] Nonetheless, due to the long latency periods, the hypothesized multistage nature of many cancers and the acknowledged cumulative, persistent and synergistic effects of chemicals in the environment, most scientists agree that a definitive answer is not likely to be forthcoming in the near future and the uncertainties will remain a reality of our time.

6. The Issues Summarized

1. Cancer is a devastating disease causing indescribable misery to its victims. The prognosis is dismal and attempts to improve survival have met with limited success. Although cancer rates are not skyrocketing, the numbers of people afflicted by this dreadful disease continue to rise.

2. Cancer is essentially a disorder of growth. Different cells within the body have different susceptibilities to develop cancer. Every indication is that cancer arises from a mutation in a single cell, causing it to lose its normal growth control, disturbing its normal functioning and replicating itself in this abnormal or neoplastic state. These cancer cells invade normal tissue and can spread, or metastasize, to other parts of the body to do further damage and perpetuate the process. Sometimes the cancer cells are so deformed from their original state that decisions as to their site of origin may be difficult.

   Various agents are known to be able to induce cancer. These include physical agents such as radiation as well as a large variety of different chemicals. Exposure to carcinogens may occur in the workplace, in medicines, in foods or in the general environment. Occupational cancers have, in fact, been known for centuries. Viruses and genetic factors have been shown to be of minimal importance in cancer causation.

   Cancer does not generally develop rapidly. The latency period, from time of exposure to the initiating carcinogen until the time the cancer is manifest, is affected by a number of factors including the total dose of carcinogen, the dose rate, the age at which exposure occurs, the sex of the individual, lifestyle factors such a nutrition, smoking and general health, and occasionally by hereditary factors. Once the mutation has occurred and carcinogenesis has been initiated, the factors that determine the rate at which the cancer will develop and consequently

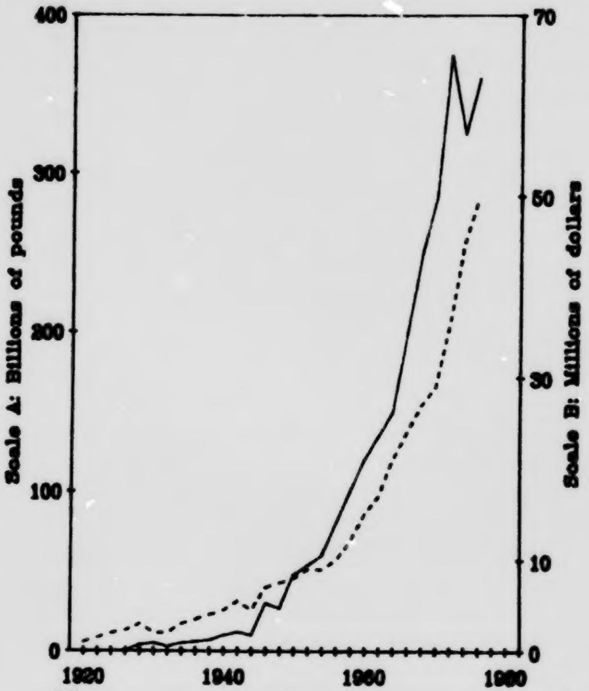

Fig. IIA-7 Annual Production All Synthetic Organic Chemicals. (excludes tar, tar crudes and primary products from petroleum and natural gas), Scale A; All chemical and allied products ...., Scale B.

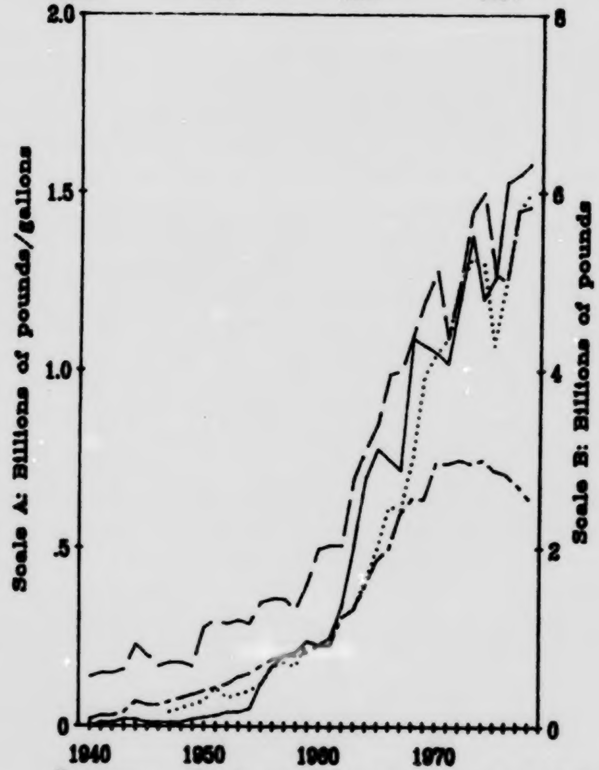

Fig. IIA-8 Annual Production of Selected Carcinogenic Substances. Acrylonitrile Scale A (pounds); Benzene --- Scale A (gallons); Perchloroethylene . - . Scale A, (gallons); Vinyl Chloride ... Scale B, (pounds).

Source: Davis and Magee, Cancer and Industrial Chemical Production, Science 206: 1356, 1979.

even if it will develop at all, have been termed "promoters". Some agents are both initiators and promoters in their own right. Epidemiological factors may affect the ability to accurately determine the biological latency period.

Other factors that determine the final outcome are the co-carcinogens or other agents present concurrently. Some substances are only carcinogenic in the presence of other substances. These latter substances may or may not be carcinogenic in their own right. Alternatively, two carcinogens may act together such that their combined action is much greater than either alone or even the added effects of the two (asbestos and smoking is the classic example).

There has been much debate as to whether or not there is a "safe dose" of carcinogens. It is generally accepted that no such threshold exists. Moreover, any additional carcinogenic exposure added to the background of carcinogens, will undoubtedly contribute to an individual's overall cancer risk. Even if the data existed to be able to determine dose-response relationships, the question as to what 'response' or what level of 'risk' would be deemed acceptable and by whom, would still be problematic.

Nonetheless, it cannot be concluded that all those who had been subject to an increased risk from an occupational exposure who developed cancer necessarily "got it from that exposure and not from another exposure" which also increased their risk. Current compensation policy therefore seems to dictate that a comparison must be made between the relative risks attributable to each exposure in isolation, especially where one is compensable and the other is not. (The difficulties inherent in this approach will be discussed at length in Part B of this chapter.)

3. Epidemiological study is the most powerful method for determining a cause and effect relationship between exposure and cancer. But it is time-consuming, costly and there are many pitfalls in epidemiological methods that must be carefully scrutinized to avoid misinterpretation of the results. A negative study does not imply that there is no increased risk, only that the study was unable to detect it, if it does indeed exist. In the final analysis it is merely a question of probabilities. There are, however, some guidelines that allow the assessment of the carcinogenic risk and several substances have thus been 'proven' as carcinogenic.

   Animal studies and chemical mutagenic studies have also contributed greatly to knowledge about carcinogenic potential. The information gained from these approaches cannot be ignored.

   Various agencies, notably the International Agency for Research on Cancer (IARC) have evaluated a number of substances for which there was both evidence of occupational exposure and data from animal and human studies regarding their carcinogenicity. Although only a small fraction of possible carcinogens has been evaluated, a list of proven and probable carcinogens has been developed. These evaluations may potentially play an important role in policy formulation in the compensation system.

4. The availability of reliable occupational health data is extremely poor. Estimates of the extent of exposure to carcinogens have been considered such a mammoth task at this point that they have not even been seriously attempted in Ontario. For the individual, the inability to provide detailed proof of exposure is a major problem in compensation battles as will be discussed in Part B of this chapter. Various efforts at improving the data bases on morbidity and mortality associated with toxic exposure are currently underway, but at a markedly slow pace.

5. Fraught with these uncertainties it is not surprising that the debate as to the fraction of cancer attributable to the workplace has been intensely bitter. Although the majority of the scientific community attribute 4-10% of present cancers to known occupational factors, the estimates range from less than 1% to greater than 40%. With the relatively recent increases in known and potential carcinogens, perhaps it is most important to stress that there may well be a significant increase in occupational cancers in years to come. Unfortunately, only time will tell; workers who are exposed to these chemicals or who have fallen victim to cancer cannot afford to wait until the uncertainties are resolved. Herein lies the challenging situation facing the compensation system.

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APPENDIX 1

Established Human Carcinogenic Agents and Circumstances, (Doll and Peto, 1981)[a]

	Exposure[b]			
Agent or Circumstance	**Occupational**	**Medical**	**Social**	**Site of Cancer**
Aflatoxin			+	Liver
Alcoholic drinks			+	Mouth, pharynx, larynx, esophagus, liver
Aklylating agents		+		Bladder, marrow
Aromatic amines	+			Bladder
Arsenic[c]	+	+		Skin, lung
Asbestos	+			Lung, pleura, peritoneum
Benzene	+			Marrow
Bis(chloromethyl) ether	+			Lung
Busulphan		+		Marrow
Cadmium[c]	+			Prostate
Chewing (betel, tobacco, lime)			+	Mouth
Chromium[c]	+			Lung
Chlornaphazine		+		Bladder
Furniture manufacture (hardwood)	+			Nasal sinuses
Immunosuppressive drugs		+		Reticuloendothelial system
Ionizing radiation[d]	+	+		Marrow and probably all other sites
Isopropyl alcohol manufacture		+		Nasal sinuses
Leather goods manufacture	+			Nasal sinuses
Mustard gas	+			Larynx, lung
Nickel[c]	+			Nasal sinuses, lung
Estrogens		+		Endometrium, vagina
Overnutrition (causing obesity)			+	Endometrium, gallbladder
Phenacetin		+		Kidney (pelvis)
Polycyclic hydrocarbons	+	+		Skin, scrotum, lung
Reproductive history			+	Breast, ovary
Parasites			+	Bladder, liver (cholangioma)
Sexual promiscuity			+	Cervix uteri
Steroids		+		Liver
Tobacco smoking			+	Mouth, pharynx, larynx, lung, emphysema, bladder
UV light	+		+	Skin, lip
Vinyl chloride	+			Liver (angiosarcoma)
Virus (hepatitis B)			+	Liver (hepatoma)

Notes

a This table is adapted from Doll and Peto, JNCI, Vol. 6, No. 6, 1981. They, in turn, expanded it from the IARC Working Group, 1980.

b A plus sign indicates that evidence of carcinogenicity was obtained.

c Certain compounds or oxidation states only.

d For example, from x-rays, thorium, some underground mining, and other occupations.

APPENDIX 2

Latent Periods in Human Occupational and Environmental Cancer

CHEMICAL SUBSTANCES

		Latent Period		
Substance	**Site**	**Range**	**Average**	**References**
4-Aminobiphenyl	Bladder	7-27	18	IARC, 1; Melick et al., 1955
Arsenic	Lung	20-	38	IARC, 2; Mabuchi et al., 1980
	Skin	5-60	18	IARC, 2; Zaldivar et al., 1981
Asbestos	Lung	c.12-	32-40	IARC, 14; Liddell, 1980; Selikoff et al., 1980
	Mesothelioma -pleura	c.17-53	40	IARC, 14; Robinson et al., 1979; Selikoff et al., 1980
	Mesothelioma -peritoneum	c.17-45+		IARC, 14; McDonald & McDonald, 1978; Selikoff et al., 1980
	G.I. Tract	c.20-		IARC, 14; Miller, 1978; Selikoff et al., 1980
	Larynx	c.17-		IARC, 14; Selikoff et al., 1980
Benzene	Leukemia	3-46	10-15	IARC, 7; Vigliani, 1976
Benzidine	Bladder	2-34	15	IARC, 1; Scott, 1962
Bis (chloromethyl) ether	Lung	8-26	13	IARC, 4; Lemen et al., 1976
Chromium and Chromium Compounds	Lung	4-47	17	Baetjer, 1950; Bidstrup & Case, 1956; IARC, 2; IARC, 23; NAS, 1974
Manufacture of Auramine	Bladder	9-28	19	IARC, 1
Manufacture of Isopropyl Alcohol	Nasal Sinus	c.10-		IARC, 15; Heuper, 1966
Melphalan	Leukemia	1-9	4	IARC, 9

Chemical Substances Continued

Substance	Site	Latent Period		References
		Range	Average	
Mustard Gas	Respiratory Tract	11-24		IARC, 9
2-Naphthylamine	Bladder	1-52	15	Case et al., 1954; IARC, 4; Scott, 1962; Uebelin & Pletscher, 1954
Nickel Refining	Lung	c.5-40+	16, 27	Morgan, 1958; NAS, 1975; NIOSH, 1977; NRCC, 1981
	Nasal Sinus	c.10-40+	22-32	Morgan, 1958; NAS, 1975; NIOSH, 1977; NRCC, 1981
Soots, Tars and Mineral Oils	Lung	10-		IARC, 3
	Skin	3-		IARC, 3
Vinyl Chloride	Liver	12-29	20	IARC, 19; Waxweiler et al., 1976
	Lung	c.15-		IARC, 19; Waxweiler et al., 1976
	Brain	c.15-		IARC, 19, Waxweiler et al., 1976
	Hemotolymphopoietic system	c.15-		IARC, 19
Wood Dust	Nasal Sinus	22-70	45	Acheson et al., 1972; Andersen et al., 1977; Engzell, 1978; IARC, 25

RADIATION

Agent	Site	Latent Period		References
		Range	Average	
Atomic Bomb (Survivors)	Leukemia	2-29+	5	Beebe et al., 1978; BEIR, 1972
	Lung	c.15-		BEIR, 1972
Radium	Bone	7-52		Rowland et al., 1978

Radiation continued

Substance	Site	Latent Period Range	Latent Period Average	References
Uranium Mining	Lung (all workers)	7-50	15-35	Archer et al., 1976; Chovil, 1981; Heuper, 1966
	Lung (non-smokers)	13-25	18	Archer et al., 1976
X-rays (therapeutic)	Leukemia	1-27	6	Court Brown & Doll, 1965

LEGEND

All values of latent periods are given in years and refer to the time from first exposure to the designated substance, process or agent until diagnosis or death, depending on the study.

c. Approximate number of years to the observed tumors: in reports containing latent periods grouped in ranges of years, the value shown is the median.

+ Indicates that a small number of cancers have been observed with latent periods exceeding the given maximum value.

IARC Refers to the designated volume of the series "IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans."

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APPENDIX 3

Evaluating Carcinogenic Risk and Known Carcinogens (IARC Methods and Findings)

A. Excerpt from IARC Monographs Vol. 1-20, Suppl. 1, 1979, p. 9-11

The data on each chemical were reviewed in detail before the meeting by two members of the group; the animal studies by an experimentalist and the human studies by an epidemiologist. Data that had become available since the publication of the relevant monograph were included in this review.

Separate assessments of the human and animal evidence of carcinogenicity were debated and adopted by the Working Group. An overall evaluation of carcinogenicity for humans was made based on the combined evidence.

Assessment of evidence for carcinogenicity from experimental animal studies.

These assessments were classified in five groups:

i. **Sufficient evidence** of carcinogenicity indicates that there is an increased incidence of malignant tumours: (a) in multiple species or strains, or (b) in multiple experiments (preferably with different routes of administration or using different dose levels), or (c) to an unusual degree with regard to incidence, site or type of tumour, or age at onset. Additional evidence may be provided by data concerning dose-response effects, as well as information on mutagenicity or chemical structure.

ii. **Limited evidence** of carcinogenicity means that the data suggest a carcinogenic effect but are limited because: (a) the studies involve a single species, strain, or experiment; or (b) the experiments are restricted by inadequate dosage levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or (c) the neoplasms produced often occur spontaneously or are difficult to classify as malignant by histological criteria alone (e.g., lung and liver tumours in mice).

iii. **Inadequate evidence** indicates that because of major qualitative or quantitative limitations, the studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect.

iv. **Negative evidence** means that within the limits of the tests used, the chemical is not carcinogenic. The number of negative studies is small, since in general, studies showing no effect are less likely to be published than those suggesting carcinogenicity.

v. **No data** indicates that data were not available to the Working Group.

The categories **sufficient evidence** and **limited evidence** refer only to the strength of the experimental evidence that these chemicals are (or are not) carcinogenic and not to the extent of their carcinogenic activity. The classification for any chemical may change as new information becomes available.

Assessment of evidence for carcinogenicity from human studies.

Evidence of carcinogenicity from human studies comes from three main sources:

1. Case reports of individual cancer patients who were exposed to the chemical or process.

2. Descriptive epidemiological studies in which the incidence of cancer in human populations was found to vary spatially or temporally with exposure to the agents.

3. Analytical epidemiological (case-control and cohort) studies in which individual exposure to the chemical or group of chemicals was found to be associated with an increased risk of cancer.

Three criteria must be met for a causal association to be inferred between exposure and human cancer.

1. There is no identified bias which could explain the association.

2. The possibility of confounding has been considered and ruled out as explaining the association.

3. The association is unlikely to be due to chance.

In general, although a single study may be indicative of a cause-effect relationship, confidence in inferring a causal association is increased when several independent studies are concordant in showing the association, when the association is strong, when there is a dose-response relationship, or when a reduction in exposure is followed by a reduction in the incidence of cancer.

The degrees of evidence for carcinogenicity in human studies were categorized as:

i. **Sufficient evidence** of carcinogenicity indicates a causal association between exposure and human cancer.

ii. **Limited evidence** of carcinogenicity indicates a possible carcinogenic effect in humans, although the data are not sufficient to demonstrate a causal association.

iii. **Inadequate evidence** of carcinogenicity indicates that the data are qualitatively or quantitatively insufficient to allow any conclusion regarding carcinogenicity for humans.

Dividing lines were by no means firmly drawn between **sufficient evidence** and **limited evidence** from animal studies and between **inadequate evidence** and **limited evidence** from both human and animal studies. When differences of opinion occurred among the members of the Working Group, the classification was made by majority vote.

Evaluation of the carcinogenic risk to humans.

Presently, no objective criteria exist to interpret the animal data directly in terms of human risk. Thus, in the absence of **sufficient evidence** from human studies, evaluation of the carcinogenic risk to humans was based on consideration of both the epidemiological and experimental evidence. Furthermore, the breadth of the categories for human and animal evidence defined above allows substantial variation within each, and the decisions reached by the group regarding overall risk incorporated these differences, even though they could not always be adequately reflected in the placement of a chemical into a particular category in the Table 3. The evidence in support of these decisions is summarized in the notes for each chemical in this Appendix.

The chemicals, group of chemicals, or industrial processes were placed into one of three groups:

Group 1

The chemical, group of chemicals, or industrial process is carcinogenic for humans. This category was used only when there was **sufficient evidence** to support a causal association between the exposure and cancer.

Group 2

The chemical or group of chemicals is probably carcinogenic for humans. This category includes chemicals for which the evidence of human carcinogenicity is almost "sufficient" as well as chemicals for which it is only suggestive. To reflect this range, this category has been divided into higher (group A) or lower (group B) degrees of evidence. The data from experimental animal studies played an important role in assigning chemicals to category 2, and particularly to those in group B.

Group 3

The chemical or group of chemicals cannot be classified as to its carcinogenicity for humans.

Table A

Organic Substances for Which There is Evidence of Carcinogenicity to Man Derived from Occupational Settings^{a,b}

Substance^b	Category of Evidence and Site^c Sufficient	Limited	Inadequate	Occupation^d
1. **Aromatic hydrocarbons**				
Coal soot Coal tar Other products of combustion	Lung, skin scrotum, bladder	larynx, kidney		Gashouse workers; asphalt, coal tar, and pitch workers; coke-oven workers; miners; still cleaners; chimney sweeps;
Petroleum Petroleum coke Wax Creosote Anthracene Paraffin Shale oils Mineral oils Soots, tars and oils	Lung, skin, scrotum	nasal cavity, larynx		Contact with lubricating, cooling or fuel with paraffin, wax, coke or rubber fillers; retortmen; textile weavers; diesel and jet testers
Benzene	Hematopoietic tissues (leukemia)			Explosives, rubber cement workers; distillers, dye users; painters; shoemakers; furniture finishers; glue makers; linoleum makers; petrochemical workers; styrene makers.
Auramine^e Benzidine B-naphthylamine a-naphthylamine^f Magenta^e A-aminodiphenyl	bladder			Dyestuffs manufacturers and users; rubber workers (pressmen, filtermen, laborers); textile dyers; paint manufacturers.

Substance[b]	Sufficient	Limited	Inadequate	Occupations[d]
2. Alkylating agents				
Mustard gas	Lung	larynx, pharynx	trachea, nasal sinuses	Mustard gas workers
Vinyl chloride	Liver (angiosarcoma)	central nervous system, lung	lympho-hematopoietic tissues, prostate, stomach, bladder	Producers and polymer producers
Bis(chloromethyl) ether	Lung			Ion-exchange resin makers; organic chemical synthesizers; polymer makers
Chloromethyl methyl ether[f]				
3. Others				
Isopropyl oils[e]	Nasal sinuses	larynx		Producers
Wood dusts	Nasal cavity and sinuses			Lumbermen; workers with woods
Leather dusts	Nasal cavity and sinuses, bladder			Leather and shoe workers

Table B

Inorganic Substances for Which There Is Evidence of Carcinogenicity to Man from Occupational Settings

Substance[b]	Sufficient	Limited	Inadequate	Occupations[d]
I. Minerals and metals[e]				
Arsenic	Lung, skin	Liver, (angiosarcoma)	hematopoietic tissues (leukemia)	Miners; smelters; insecticide and herbicide makers and sprayers; tanners; chemical workers; oil refining workers; vintners; alloy makers; aniline color makers; ceramic makers; copper smelters; textile printers
Beryllium		Lung		Aerospace industry, gyroscopes and nuclear reactor workers; beryllium cathode ray tube makers; electric equipment makers; electronics workers
Cadium		Prostate, kidney, lung		Alloy makers; battery makers; pesticide workers; textile printers; welders; zinc refiners; electroplaters
Chromium	Lung	Nasal cavity and sinuses, larynx		Producers and processors; acetylene and aniline workers; bleachers; glass, pottery and linoleum workers; battery makers; anodizers; electroplaters; glass workers; lithographers; photoengravers
Hematite[e]	Lung		larynx	Iron ore (hematite) miners; metal grinders and polishers; iron industry workers
Nickel[e]	Lung, nasal sinuses			Nickel smelters, mixers, and roasters; electrolysis workers
Asbestos	Lung pleura and peritoneum (mesothelioma)	larynx, colon, rectum		Miners; millers; textile, insulation and shipyard workers; brake lining workers

Notes

a Source: Cole, P. and Merletti, F. Chemical Agents and Occupational Cancer, J. Env. Path and Tox. 3,4: 399, 1980, see Appendix 4, Table C for more recent evidence not included in this table.

b Restricted to chemical substances for which there is evidence of carcinogenicity to man derived from occupational settings; see Appendix 4 Table C for more recent evidence not included in this table.

c The explanation of the categories for evidence is as follows: "sufficient" - human data are considered persuasive of carcinogenicity for humans; "limited" - evidence exists for humans but is too limited to be persuasive; "inadequate" - human data have been considered qualitatively or quantitatively insufficient to allow any conclusion regarding carcinogenicity of the substance to humans.

d A partial listing of categories where exposure may have been heavy or extended to many persons.

e For auramine, magenta, isopropyl oils, hematite and nickel the evidence of carcinogenicity pertains to processes involving the substance. For minerals and metals it may be that only some compounds or industrial processes are carcinogenic.

f Carcinogenic activity may reflect contamination with the previously-listed substance.

APPENDIX 4

Table A

Proportion of Cancer Attributed to Various Different Factors by Various Authors[a]

Factor or Class of Factors	Doll and Peto '81[c]		Higginson and Muir '79[c]		Wynder and Gori '77[c]	
	Best Estimate	Range	M	F	M	F
Tobacco	30	25-40	30	7	28	8
Tobacco/alcohol	-[b]	-	5	3	4	1
Alcohol	3	2-4	-	-	-	-
Diet	35	10-70	-	-	40	57
Food Additives	1	-5 - -2	-	-	-	-
Reproductive and Sexual Behavior	7	1-13	-	-	-	-
Lifestyle	-	-	30	63	-	-
Occupation	4	2-8	6	2	4	2
Pollution	2	1-5	-	-	-	-
Industrial Products	1	1-2	-	-	-	-
Medicines and Medical Procedures	1	0.5-3	1	1	-	4
Geophysical Factors[c]	3	2-4	11	11	8	8
Infection	10	1-	-	-	-	-
Congenital	-	-	2	2	-	-
Congenital/Unknown	-	-	-	-	16	20
Unknown			15	11	-	-

Notes

a Source: derived from tables in Doll and Peto, 1981, op. cit.
b Various authors classified the factors differently.
c Doll and Peto referred only to cancer deaths, the other authors referred to cancer cases. Cancers from UV light may account for a much greater proportion of cancer cases than cancer deaths (non-fatal skin cancer).

Table B

Doll and Peto's Classification of Cancers by Evidence of Work-Relatedness[a]

---

a. Cancers that are not **known** to be produced by occupational hazards:

lip
tongue
pharynx
small intestines
gallbladder and bile ducts
melanoma
breast
cervix
uterine
ovary and other female genital organs
male genital organs other than prostate
eye
thyroid
myeloma

b. Cancers that **possibly** may be produced by occupational hazards:

mouth
esophagus
stomach
colon and rectum
pancreas
connective tissue
kidney
brain
Hodgkin's disease
non-Hodgkins lymphoma

c. Cancers that **definitely** can be produced by occupational hazards:

mesentery and peritoneum (M: 15%, F: 5%)[b]
liver and intraphepatic bile ducts (M: 4%, F: 1%)
larynx (M: 2%, F: 1%)
lung (M: 15%, F: 5%)
pleura, nasal sinus and remaining respiratory sites (M: 25%, F: 5%)
bone (M: 4%, F: 1%)
skin other than melanoma (M: 10%, F: 2%)
prostate (M: 1%)
bladder (M: 10%, F: 5%)
leukemia (M: 10%, F: 5%)
other and unspecified (M: 6.8%, F: 1.2%)

Notes

[a] Source: Doll and Peto, JNCI 66: 1191, 1981.
[b] Numbers in parentheses represent the percent of cancer deaths of that site ascribed to occupational hazards in males and females respectively.

Table C

Recent Epidemiological Evidence Suggestive of a Relationship between Occupation and Cancer by Site[a]

Cancer Site	Occupation, Industry, Exposure	Reference
Prostate	rubber and tire workers	Goldsmith et al., 1980
	cadmium exposure	Cole & Merletti, 1980
	cadmium production workers (248)[b]	Lemen et al., 1976
Colon and Rectum	paint and coating manufacturing	Morgan et al., 1980
	coke by-product workers (181)[b]	Redmond et al., 1976
	smelter and battery-plant workers	Kang et al., 1980
Stomach	petroleum refining and petrochemical plants	Thomas et al., 1980
	ethylene oxide, ethylene dichloride	Hogstedt et al., 1979
	coal miners (40)[b]	Rockett, 1977
	rubber industry-processing (80)[b]	Monson & Nakano, 1976
Brain	petroleum refining and petrochemical plants	Thomas et al., 1980
	rubber industry-tire building (90)[b]	Alexander et al., 1981 Monson & Nakano, 1976
Leukemia	petroleum refining and petrochemical plants	Thomas et al., 1980
	ethylene oxide, ethylene dichloride	Hogstedt et al., 1979
	rubber workers	Wolfe et al., 1981
	rubber industry-processing (140)[b]	Monson & Nakano, 1976
	shoe workers (100)[b]	Aksoy et al., 1974
Multiple Myeloma	petroleum refining and petrochemical plants	Thomas et al., 1980
Respiratory System	welders,shopfitters and metal-trades workers	Beaumont & Weiss, 1980
	plumbers and pipefitters	Kaminski et al., 1980
	arsenic-exposed workers	Pinto et al., 1978
Lung	aluminum reduction plant workers	Milham, 1979
	laundry and dry cleaning workers	Blair et al., 1979
	spray painters, zinc chromate primer paints	Delagar et al., 1980
	beryllium, cadmium	Cole & Merletti, 1980
	automobile manufacture (casting and plating)	Silverstein et al., 1981
	foundry workers (50-150)[b]	Koskela et al., 1976
	metal miners (200)[b]	Gibson et al., 1977 Wagoner et al., 1963

Cancer Site	Occupation, Industry, Exposure	Reference
	rubber industry-tire curing (61)[b]	Monson & Nakano, 1976
	smelter and battery plant workers	
Soft Tissue Sarcomas	phenoxyacetic acids and chlorophenols	Hordell & Sandstom, 1979
Lymphoma	aluminum reduction plant workers	Milham, 1979
	chemists (79)[b]	Li et al., 1969
Lymphatic-hematopoietic		
Pancreas	plumbers and pipefitters	Kaminski et al., 1980
	aluminum reduction plant workers	Milham, 1979
	chemists (64)[b]	Li et al., 1969
	coke by-product workers (312)[b]	Redmond et al., 1976
Esophagus	plumbers and pipefitters	Kaminski et al., 1980
Kidney	female laundry and dry cleaning workers	Katz & Jowett, 1981
	cadmium	Cole & Merletti, 1980
Uterine-cervical and other genital cancers	female laundry and dry cleaning workers	Katz & Joewtt, 1981
		Blair et al., 1979
Bladder	leather and leather products manufacturing (150)[b]	Cole & Goldman, 1975
		Decoufle, 1979
	rubber industry-tire building (88)[b]	Monson & Nakano, 1976
Mouth and Pharynx	textile workers (77)[b]	Moss & Lee, 1974
	printing pressmen (125)[b]	Lloyd et al., 1979
	newsprint pressroom workers	Nicholson et al., 1981
Nasal Cavity and Sinuses	furniture workers (300-400)[b]	Acheson, 1976
	shoe workers (700)[b]	Acheson, 1976
		Brinton, 1977

Notes

[a] Source: Davis et al, Banbury Report No. 9, 1981, table 2. (Complete reference for each of these studies is provided.)

[b] These data are derived from a table of occupational groups in which excess cancer incidence has been reported without identification of a specific etiological agent. The numbers in parentheses refer to percent excess reported. Source - Davis et al, 1981, op. cit. Table 3. (Complete reference for each of these studies is provided.)

(Although less recent, these studies are included here as substantiating studies; also, these links had not previously been widely-recognized.)

Commentary:

It is of interest to note that there are many cancer sites on the above list which according to Doll and Peto (Table B) were not known to be produced by occupational hazards, e.g. myeloma, uterine, cervical and other genital cancers. Others on this list, e.g. kidney, pancreas, colon and rectum, pancreas, stomach, esophagus, mouth, brain and lymphoma, were only considered by Doll and Peto as possibly produced by occupational hazards, for which they allocated a token 1% of male cancer deaths and 0.5% of female cancer deaths. The validity of this allocation is largely determined by the numbers of workers in each of the occupations or industries or subject to the exposures listed in Table C (as well as other well-recognized carcinogenic exposures) and the intensity of the exposure.

B. OCCUPATIONAL CANCER AND THE ONTARIO WORKERS' COMPENSATION SYSTEM

1. Historical Role and the Development of Guidelines

1.1 Historical Role Played by the Ontario Workers' Compensation Board

The prolonged latency period that characterizes cancer, the lack of accurate exposure information, the dearth of research in the field, the reality of multifactorial etiology and the other issues discussed above make it difficult to establish with any great certainty the validity or work-relatedness of any individual cancer claim (with the exception of some rare types of cancer, such as mesothelioma and angiosarcoma). Thousands die each year or are disabled by cancer, the vast majority of whom are either currently employed or were at an earlier date, often in conditions of considerable exposure to dusts or fumes. Meanwhile the list of proven or suspected carcinogens in the workplace continues to grow. As discussed above, even if stringent regulations were currently enforced, the problem posed by cancer for workers' compensation systems is potentially staggering and will continue to be so for some years to come.

The Ontario Workers' Compensation Board has played an historical role in the face of massive concerns regarding occupational cancer. Its record as compared to other jurisdictions is impressive, both in terms of policy development and actual numbers of cases being compensated (as shown in Chapter I). The Ontario Board was the first compensation jurisdiction to recognize and compensate cancer among foundry workers, and laryngeal and gastrointestinal cancer among workers exposed to asbestos. In 1950, the first case of occupational lung cancer was accepted (coal tar exposure), becoming one of the first of its kind in any compensation agency in Europe or North America.[1] To date, nine sets of cancer guidelines have been developed as shown in Table IIB-1.

The Ontario Board was also the first jurisdiction in Canada to compensate asbestos-related lung cancer in the absence of clinical asbestosis.[2] Even today, the British Columbia schedule of diseases, for example, stipulates that a claimant must suffer from asbestosis in order for the statutory presumption in favour of compensating lung, laryngeal or pharyngeal cancer to be invoked. However, neither time nor quantitative exposure requirements are imposed. A similar statutory presumption also exists for compensating gastrointestinal cancers but only where there has been a period of cumulative, continuous exposure to asbestos dust of 20 years and the exposure represents a major component of the worker's activity. (See Appendix 1.) Quebec also has a statutory presumption for compensating asbestos-related lung cancers, but only in the presence of asbestosis. Quebec compensates mesothelioma, as well, but it has not yet awarded compensation for asbestos-related cancers of the pharynx, larynx or the gastro-intestinal system.

In the schedule of diseases used in British Columbia, over ten different types of cancer are compensable from exposure to a variety of different carcinogens as shown in Appendix 1. However, only a handful of cancer claims have been compensated (as shown in Chapter I). In Newfoundland, the schedule of diseases includes "cancer, lung cancer, stomach cancer, and carcinoma or malignant disease arising from radiation". However, no

Table IIB-1

Historical Development of the WCBO Policy With Respect to Cancer

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1914 - The Workmen's Compensation Act of Ontario was passed

1932 - Schedule 3 of the Act was amended to include: Cancer - arising from the manufacture of pitch and tar

1940 - Item 9 was added to Schedule 3: "any disease due to exposure to X-rays, radium or other radioactive substances."

1942 - Schedule 3 of the Act was further amended to specify that only cancer of the skin due to pitch and tar was intended and only in certain processes:

Description of Disease	Description of Process
Epitheliomatous cancer or ulceration of the skin due to tar, pitch, bitumen, mineral oil or parrafin, or any compound, product or residue of any of these substances.	Handling or use of tar, pitch, bitumen, mineral oil or paraffin or any compound, product or residue of any of these substances.

1947 - General clause covering industrial diseases became effective

1949 - 1st Guidelines passed re: lung cancer in coal gasification workers

1950 - 1st allowed cancer claim (lung cancer in coal gasification workers)

1956 - Guidelines passed re: lung cancer in arsenic exposure

1961 - Guidelines passed re: lung and sinus cancer in nickel refining (revised 1961, 1972 and 1981)

1976 - Guidelines passed re: lung cancer in coke oven exposure (revised 1976 and 1978)

- Guidelines passed re: lung cancer in uranium mining (revised 1979)
- Guidelines passed re: lung cancer and mesothelioma in asbestos exposure
- Guidelines passed re: gastrointestinal malignancy in asbestos exposure

1978 - Guidelines passed re: larynygeal cancer - asbestos and nickel exposure (proposed revision 1981)

1979 - Guidelines passed re: Foundry exposure (revised 1981) cancers from outside the fluorspar mines have yet been compensated. In Saskatchewan, which does not use a schedule of diseases at all but adjudicates each case on its own merit, the few dozen claims compensated are still unimpressive. Manitoba, which also does not use a schedule of diseases, does have guidelines for the allowance of cancer of the larynx and gastrointestinal cancer in asbestos exposure, with specific requirements in terms of duration of exposure for the former and inception period requirements for both. A minimum of 20 years inception period is required for gastrointestinal cancer from asbestos exposure; 15 years exposure to nickel aerosols, 10 years exposure to asbestos or 7.5 years nickel as well as 5 years asbestos exposure in the case of laryngeal cancer in dual exposure.[5] As will be shown in Section 3, Ontario's requirements for compensation seem to compare favourably to other jurisdictions, as do its statistics discussed earlier.

1.2 The Development of Policy

The Workmen's Compensation Act came into effect in 1915. As alluded to in Chapter I, under section 1(1)(n) an "industrial disease" is defined as" any of the diseases mentioned in Schedule 3, and any other disease peculiar to or characteristic of a particular industrial process, trade of occupation." Section 122 creates a rebuttable presumption in favour of compensating a worker who suffers from a disease named in Schedule III of the Act if the worker was employed in a occupation, trade or process deemed by the statute to have caused it.

On March 31, 1947, a provision was added to the Act giving the Workers' Compensation Board the right to designate diseases as "peculiar to or characteristic of ..." by regulation. (Some reports state that the recognition of malignancies as work-related actually dates back to an amendment of the statute in 1932.) There are therefore three approaches to the adjudication of cancer claims. First, the disease can be included in the schedule along with the process or exposure. (This is the situation for skin cancer). Secondly, the Workers' Compensation Board can approach each claim as if it were new. (A few cases of cancer have been awarded compensation on their own merit). Thirdly, the Board may choose to adopt a policy to guide the allowance or rejection of the claim subject to certain characteristics of the circumstances. (The vast majority of cancer claims at the Ontario Workers' Compensation Board have been adjudicated by this method.)

The stated purpose of the guidelines, which are developed by the Medical Services Division in conjunction with the Claims Services Division, is to make adjudication uniform. The guidelines define the minimum requirement that must be met to clearly establish that the cancer is indeed due to workplace conditions with the provision that claims which do not meet the guidelines may be individually judged on their own merit. The Board states that if a claim is the first of its kind received, or if the Board receives a number of claims from workers in the same plant or industry, a decision may be made to conduct studies into the cause-effect relationship and the need for adjudication guidelines. Sources usually involved include the Industrial Disease and Dependents Section of the Claims Adjudication Branch, the Medical Services Division, as well as outside scientists and other experts as chosen by the Board. The Medical Services Division conducts or supervises the research although it is their opinion that this is the responsibility of the Ministry of Labour's

Occupational Health Branch. Previously much of this research was carried out by the Ontario Ministry of Health. Now, it is usually done by university staff or by research agencies such as the National Cancer Institute of Canada (NCIC) or by the Board itself.

The search may begin with a literature review (often via terminals at the Ontario Ministry of Labour, or more recently at the Industrial Accident Prevention Association (IAPA)). If the focus is on one plant, or the process is initiated by a specific claim or group of claims, the Claims Services Division may endeavour to conduct a field study, often difficult as the current conditions may not be representative of twenty years earlier, or possibly the plant may not still be in existence. As is the procedure in most cancer claims, the claims investigator would question the worker (or workers, in the case of multiple claims) or the spouses if the workers are deceased, as well as current and former co-workers. Past conditions in the workplace are discussed, as well as personal habits outside the workplace of the workers in question. Management, foremen, the safety officer and the plant doctor may be interviewed by the claims investigator. Company records of employees, substances purchased or produced and manufacturing procedures are studied where they exist, with particular attention to conditions in the area where the worker had worked. Relevant data from the files of the Occupational Health Branch of the Ministry of Labour may also be sought.

Information from the literature review and from the field study may be deemed by the Board as sufficient for the medical division to prepare initial guidelines to be examined by the claims and medical service staff and finally submitted to the corporate board for approval. Often, however, it is decided that an epidemiological study is needed to determine if there is indeed a significantly higher incidence of the disease in question, among a certain work force. As was discussed in Part A of this chapter, this is usually a long painstaking endeavour. Guidelines may be reviewed and amended as new scientific information emerges or for less strictly scientific reasons, as will be shown in Section 3.

The historical role played by the Ontario Workers' Compensation Board has been internationally commended and has served as a model for many other jurisdictions. The road, however, has been plagued with controversy. Although the formulation of guidelines establishing that certain cancers are work-related has been favourably received, the specific circumstances of allowance or rejection have been the source of contention from labour's point of view as needlessly restrictive and even contrary to scientific knowledge. Certain sectors of management have criticized the guidelines as overly generous. Since the guidelines have the potential for establishing which claims will be allowed and which will not, the procedure by which they are developed takes on considerable importance.

The development of guidelines for the various cancers now recognized by the Ontario Workers' Compensation Board was carefully reviewed for this Inquiry. Certain points are worth noting, some of which were alluded to by Barth.[4] First, the development of guidelines has been largely internal to the Board, with only two outsiders playing prominent roles, Dr. Ritchie, pathologist from the University of Toronto, and Dr. Miller of the NCI. Secondly, even when outside opinions are solicited, they may be ignored. Thirdly, few persons outside the Workers' Compensation Board are aware that the process is taking place, and although the relevant employers are occasionally consulted, labour organizations rarely, if ever, have been (at least until recently). Fourthly, the process of developing guidelines has generally been slow and fraught with delays. Although in one instance, guidelines were set prior to the completion of a study, resulting in some embarrassment, generally haste does not characterize the process. Finally, and perhaps most importantly, there is no system that initiates the development of new guidelines. Many insist that the Board's initiatives have merely been the culmination of years of hard struggle waged by working people in Ontario. The process seems to be triggered largely by political pressure hence the evaluation of guidelines tends to lag considerably behind the existing scientific knowledge. As the guidelines play such a paramount role in the process of adjudicating cancer claims, these procedural issues will be discussed in depth in Section 3, along with the assumptions that underlie the content of the guidelines.

What has emerged is that there is a strong feeling that many who deserve compensation are not receiving it. As will be shown in the following section, the problem lies not merely in the guidelines and the adjudication process, but also in the recognition and reporting of occupational cancer. Recommendations with respect to both aspects of this situation are warranted and will be discussed below.

2. Facts, Figures and Issues

2.1 The Volume of Cancer Claims

Although cancer is a leading cause of death in Ontario and accounted for the death of 14,252 Ontario residents in 1979,[5] cancer claims represent but a tiny percentage of the workload of the Ontario Workers' Compensation Board.

As shown in Chapter I the allowed industrial disease claims in Ontario are annually in the range of 1.1% to 1.9% of all compensated claims. Cancer claims consistently represent less than 1% (ranging from .32% to .84%) of the total disease claims and less than 0.016% of total allowed claims. Even excluding "medical aid only" claims, cancer claims represent only slightly more than 1% of industrial disease claims and 0.04% of the total allowed claims as shown in Table IIB-2a. Although cancer claims have risen slightly on a proportional basis, they still remain only a small percentage of all "industrial disease" claims and an even more trivial fraction of all claims processed.

It should be noted, however, that cancer does represent a significant proportion of the fatal claims allowed at the Workers' Compensation Board, (Table IIB-2b) representing over 10% of all compensated fatality claims in Ontario over the last 3 years. The number of cancer claims filed constitutes an even a higher percentage of the total fatal claims reported, as shown in Table IIB-3, accounting for 18.6% of all fatalities reported to the Board in 1980. Thus, although the number of claims is very small, the almost inevitably fatal outcome gives this small group of claims immense importance.

Table IIB-2a

All Allowed Compensation[a] Claims, Industrial Disease Compensation Claims and Cancer Claims, 1974 - 1980[b]

Year	All Claims[a]	Industrial Disease Claims	% of Total	Cancer Claims	% of Industrial Disease Claims	% of Total
1980	165,221	5,905	3.6	64	1.1	0.04
1979	165,456	5,899	3.6	66	1.1	0.04
1978	150,385	5,702	3.8	41	0.7	0.03
1977	149,213	6,015	4.0	50	0.8	0.03
1976	154,910	5,766	3.7	38	0.7	0.02
1975	141,186	5,061	3.6	16	0.3	0.01
1974	149,204	4,774	3.2	n.a	n.a	n.a

Notes

a Claims referred to in this table are "1st Payment Compensation Claims," i.e. exclude "medical aid only" claims.

b Source: Ontario Workers' Compensation Board, Annual Report, 1981, Appendix A.

Table IIB-2b

All Allowed Fatal Claims, Fatal Disease Claims and Fatal Cancer Claims[a]

Year	All Fatal Claims	Fatal Industrial Disease Claims	% of All Fatal Claims	Fatal Cancer Claims	% of Fatal Industrial Disease Claims	% of Total Fatal Claims
1980	324	94	29	42	45	13
1979	323	119	37	33	28	10
1978	261	84	32	29	35	11

a Source: Figures cited in Peter S. Barth. Workers' Compensation and Asbestos in Ontario. A study for the Dupre Commission; Adapted from WCB tables.

Table IIB-3

All Fatal Claims Reported in 1980 by Claim Status and Cause of Death[a]

Listed Cause of Death	Claims Allowed		Rejected				
	Old[c]	New	Old	New	N.F.A.[b]	Pending	Total
Accidental Death[d]	24	166	23	23	32	5	273
100% P.D.[e]	63	-	-	-	-	-	63
Disease:							
Cancer	25	19	29	29	2	4	108
	(44)[f]		(58)				
Cardiac disease	14	4	28	35	1	-	82
Respiratory disease	9	2	13	7	-	-	31
Hepatic failure	-	-	1	1	1	-	3
Pancreatitis	-	-	1	-	-	-	1
Renal Failure	-	-	2	-	-	-	2
Gastrointestinal disease	1	-	1	-	-	-	2
Misc. or unspecified	1	3	2	8	-	-	4
Total	50	28	77	80	4	4	243
	(78)		(157)				
Grand Totals	**137**	**194**	**100**	**103**	**36**	**9**	**579**
	(331)		(203)				

Notes

a. This table was derived from the Fatal Claims Record maintained by the Industrial Disease and Dependents Section. The Cause of death was listed by a coder from information provided in the claim file, usually from the death certificate.

b. No Further Action.

c. "Old" refers to fatal claims filed for workers already receiving benefits (e.g., a worker receiving a large pension for silicosis, for example, who dies of cancer, may be included in this list without a "cancer claim" necessarily being recorded for purposes of Table IIB-2a).
"New" refers to claims set-up upon death of the claimant.

d. Accidental death includes all claims where cause of death was listed as falls, crushing, asphyxiation, electrocution, drowning, burn, murder, motor vehicle accident, etc.

e. Those workers receiving 100% permanent disability benefits at the time of their death receive automatic fatal benefits for their survivors. Cause of death is therefore not listed. Other sources indicate that 26 of the 63 claimants on 100% PD died of causes not related to their compensable condition.

f. Total allowed and rejected claims are included in parentheses. Note that cancer claims make up 18.6% of the total fatal claims filed and 44% of the disease claims constituting 13.3% of the allowed fatal claims and over 56% of the allowed fatal industrial disease claims.

(The reader may note that there are many discrepancies in the Ontario Workers' Compensation Board statistics used in the various tables in this report. Different figures come from different sources within the Board, e.g. Industrial Disease and Dependents Section of the Adjudication Branch provides one set of statistics, the Program Planning and Statistical Services Branch provides another, the Medical Services Division provides yet another, and those derived personally from counting up claims, for example, in the fatal claims record, provide yet a fourth source. No explanation is provided by Barth as to his sources of data. Despite many attempts at rationalizing these differences, no clear explanation is forthcoming. The statistics provided by the Program Planning and Statistical Services Branch are the "official" figures. The reader is referred to the notes at the bottom of each table as to how the particular statistics were derived.)

2.2 *Rejection Rate of Occupational Cancer*

Table IIB-3 also illustrates that whereas 16.8% of the fatal accident claims reported in 1980 were rejected, 53.7% of the fatal cancer claims were rejected (see Table IIB-4b for claim status by cancer site). Looking at the non-fatal claims, the discrepancy between the rejection rate for claims overall (96.7% of which are accidents) and that for cancer claims is even greater. According to the records of the Industrial Disease and Dependents Section of the Claims Adjudication Branch, as shown in Table IIB-4, in 1980 63.8% of the cancer claims adjudicated were rejected (as were 58.7% of those adjudicated in 1981). These figures were consistent with the findings of the industrial disease survey performed for this Inquiry by the Program Planning and Statistical Service Branch of the Board. As shown in Table IIB-6, 32 of the 105 claims reviewed were allowed, 54 were rejected and 19 were withdrawn or required no further action. Thus, the rejection rate for cancer claims in this survey was 62.7% of those adjudicated.

In contrast, as shown in Table IIB-6, the rejection rate for claims overall was 6.4% in 1980. Excluding claims that were rejected for administrative reasons, the rejection rate was only 1.1%. Thus, while only about 1% of accident claims are denied, based on their merits, almost one-third of cancer claims are denied. While this discrepancy may not be astounding if one considers the complexity of adjudicating cancer claims in comparison with accident claims, it certainly highlights the very different nature of the two.

2.3 *Rejection Rate and Guidelines*

Records of cancer claims are kept by the medical staff of the Workers' Compensation Board for all cancer claims that are addressed by guidelines. As shown in the upper left portion of Table IIB-7, of 698 cancer claims received in this category (to end of 1981), 535 were allowed and 154 were rejected. By cancer site, this represents the following: 403 lung cancer claims were allowed compared to 104 rejected, 57 sinus cancer claims were allowed and 4 rejected, 51 mesothelioma claims were allowed and 6 rejected, 9 cases of gastrointestinal cancer were allowed and 17 rejected and 15 laryngeal cancer claims were allowed and 23 rejected; nine claims were pending. On this basis, the Board claims to allow 78% of its cancer claims, rejecting a mere 22%. However, the rejected claims in this portion of the table represent only those claims for cancer of a specific site recognized by the Board with proven exposure to the appropriate carcinogen listed. Claims for gastrointestinal cancer in coke oven workers, for example, have not been reported for a number of years, nor have laryngeal cancers in uranium miners, and so on.

The remainder of the table shows that 43 cases of skin cancer had been allowed, 5 cases of bladder cancer, 4 cases of leukemia, and 1 other. These latter cancer sites are not adjudicated by guidelines. Skin cancer is a scheduled disease under the Workers' Compensation Act. The five bladder cancer claims and four leukemia claims were the only ones to have been allowed by the Board in the absence of guidelines (excluding a few precedent-setters that were allowed prior to approval of guidelines in their respective categories). The very few cases to have been allowed based on their own merits itself attests to the importance of the guidelines.

Data are not readily available concerning the rejected claims in the last three cancer sites mentioned, or for cancers where the exposure to a specific agent is either not proven, not recognized by Board guidelines, or not of a site mentioned in the guidelines. Thus, the actual rejection rate is not reflected in this tabulation.

Records of all cancer claims reported to the Board are maintained by the Industrial Disease and Dependents Section of the Claims Adjudication Branch, with the cancer site and exposure in question listed as shown in Table IIB-8. These, however, are not coded by allowed or rejected status. From this table it can be seen that at most 79% of the 95 claims reported in 1980, and 40 of the 60 in 1981 would be adjudicated by guidelines. This would imply that possibly one-third of the cancer claims filed would have to be judged on their own merit and would therefore be more likely to be rejected, due to the greater burden of proof applicable. By dividing the cancer claims surveyed for this Inquiry by exposure as shown in Table IIB-9 it is evident that all claims for cancer resulting from exposures not covered by guidelines were indeed rejected. (These represented at least one-quarter of the claims. Not all the rejections for cancers in which exposures are covered by guidelines would be included in the "official rejections" as many are of the wrong cancer site as illustrated in Section 3 of this chapter.)

It has often been stated by the Board that comparing allowed to rejected rates is misleading as the reasons for rejections are numerous and varied. These reasons include: (1) incorrect diagnosis (i.e. not really cancer), (2) lack of scientific evidence that cancer is work-related, (3) inadequate exposure to carcinogen in question, inadequate latency period, excessive cessation interval or inappropriate tumour type with respect to exposure in question, or (4) technical reasons including out-of-province exposure or claim not substantiated with appropriate documentation either from the claimant or from the company in question or possibly through medical reports.[6]

In the vast majority of cancer claims in the Industrial Disease Survey, personally reviewed, it was clear that reasons 1 and 4 did not account for a significant number of rejections.

Even excluding claims rejected for these reasons, it seems that the rejection rate for cancer claims is over 60% and is significantly higher than the rejection rate for claims overall.

Table IIB-4a

Cancer Claims Reported to the WCBO in 1980 (and 1981)[a]

by Claim Status and Cancer Type

Cancer Type	No. Reported to WCB	Allowed	Denied	Pending
Lung	68 (33)	14 (4)	28 (14)	26 (15)
Mesothelioma	10 (3)	4 (1)	3 (0)	3 (2)
Sinus	4 (3)	2 (2)	1 (0)	1 (1)
Throat	2 (10)	0 (3)	1 (2)	1 (5)
G.I.	7 (6)	0 (1)	5 (1)	2 (4)
Bladder	1 (1)	0 (0)	0 (0)	1 (1)
Skin	2 (3)	0 (0)	0 (0)	2 (3)
Leukemia	1 (1)	0 (0)	0 (0)	1 (0)
Total	**95 (60)**	**20 (12)**	**37 (17)**	**37 (31)**

Source: Industrial Disease and Dependents Section of the Claims Adjudication Branch.

a - parentheses refer to 1981 figures.

Table IIB-4b

Fatal Cancer Claims Reported in 1980

by Claim Status and Cancer Type[a]

Cancer Type	No. Reported to WCB	Allowed		Denied		Pending
		Old	New	Old	New	(or no further action)
Lung	26	6	1	10	9	-
Laryngeal	2	-	1	1	-	-
Sinus	1	1	-	-	-	-
Mesothelioma	5	4	1	-	-	-
Leukemia	2	-	-	2	-	-
Prostate	1	-	-	1	-	-
G.I.	3	-	-	1	1	1
Unspecified	68	14	16	14	19	5
		25	19	29	29	
Total	**108**	**44**		**58**		**6**

Notes

a. This table was derived from the Fatal Claims Record maintained by the Industrial Disease and Dependents Section. The cause of death was listed by a coder from information provided in the claim file, usually from the death certificate.

Table IIB-5

Cancer Claims Included in the Industrial Disease Survey[a] by Last Claim Status

Last Claim Status	No. of Claims	% of Total	% of Those Adjudicated[d]
Allowed	32	30.5%	37.2%
Rejected	54	51.4%	62.8%
Claimant did not reply[b]	5	4.8%	
No action[c]	14	13.3%	
Total	**105**	**100.0%**	**100.0%**

Notes

a. This survey, conducted by the Program Planning and Statistical Services Branch for this Inquiry, includes only claims set up prior to July 1, 1979. Claims were reviewed in reverse chronology from that date. (The date was chosen to allow ample time for the claim to have moved through all the different stages of adjudication.)

b. Claimant failed to reply to request for evidence and claim was dropped.

c. These largely consisted of claims which were set-up due to excessive exposure to a known carcinogen. (Specifically, in this case, exposure was due to a malfunction), however, no disability had yet resulted hence the claims have been inactive.

d. Total excludes the 19 claims which were not adjudicated (see b & c).

Table IIB-6

Total Rejected Claims 1979, 1980 by Category of Rejection[a]

	1979		1980	
Total Claims	460,972		444,674	
Category 1 Entitlement Rejections	6,196	(1.3%)	4,673	(1.1%)
(Employee has a right to make a claim)				
Category 2 Administrative Rejections	29,366	(6.4%)	23,659	(5.3%)
(Claim should not have been submitted)[b]				
Total Rejections	**35,562**	**(7.7%)**	**28,332**	**(6.4%)**

[a] Source: Program Planning and Statistical Services, WCBO

[b] These include claims filed for accidents occurring outside Ontario, or in businesses not covered by the Act, or claims filed by executive officers of companies. Also included are claims where the employer has no record of the claimant's employment; and claims rejected for other administrative reasons.

Table IIB-7

Allowed (and Rejected)^a Cancers by Site and Exposure Substance - Total to December 1981

	Lung	Sinus	Mesothelioma	G.I.	Laryngeal	Skin	Bladder	Leukemia	Other	Total
Nickel	165 (20)	57 (2)			10 (14)					
Radiation	85 (40)	- (2)						3		
Asbestos	59 (10)		51 (6)	9 (11)	5 (9)					
Arsenic	33 (12)									
PAH										
-(gas co.	12 (-)									
-coke ovens)	16 (9)	-		(6)						
Foundry										
- Hamilton	27 (9)									
- Other	6 (4)									
	403 (104)	57 (4)	51 (6)	9 (17)	15 (23)					535 (154)
Aniline dye							5			
Benzene								1		
Unspecified						43			1	
Total	**403**	**57**	**51**	**9**	**15**	**43**	**5**	**4**	**1**	**588**

Notes

a. Rejected claims are depicted in parentheses; nine pending claims are not shown. The top left portion of the table represents claims adjudicated by guidelines, and rejections are only routinely enumerated for claims that are specifically addressed by the guidelines. As gastrointestinal (G.I.) cancer in coke oven workers is not addressed by the guidelines, no allowances have been made. Moreover, the 6 rejected claims do not represent all G.I. cancer claims from coke oven workers that were rejected. Apparently these were recorded before the decision was made not to record these cases. No rejection figures are available for the remainder of the table, nor for cancer sites that do not correspond to the specified exposures (e.g. no figure is available for the number of rejected lung cancer claims from workers exposed to chromates, etc., nor is the number of rejected G.I. cancer claims from foundry workers noted.)

Table IIB-8

Cancers Reported by Site and Exposure Substance[a] - 1980 and (1981)[b]

	Uranium	Coke Oven	Foundry	Nickel	Arsenic	Asbestos	Other	Total
Lung	13 (4)*	2 (-)*	11 (4)*	12 (15)*	4 (2)*	21 (4)*	4 (4)	68 (33)
Mesothelioma						10 (3)*		10 (3)
Sinus				4 (3)*				4 (3)
Throat			- (1)	1 (4)*		1 (1)*	- (4)	2 (10)
G.I.	1 (-)	1 (-)	- (3)	3 (2)			2 (1)	7 (6)
Bladder						1	- (1)	1 (1)
Skin						2 (-)	- (3)	2 (3)
Leukemia	- (1)						1 (-)	1 (1)
Total	**14 (5)**	**3 (0)**	**11 (8)**	**20 (24)**	**4 (2)**	**35 (8)**	**7 (13)**	**95 (60)**

Notes

a Source: Industrial Disease & Dependents Section of the Claims Adjudication Branch.

b Numbers in parentheses represent 1981 totals.

* Claims that would be adjudicated according to guidelines.

Table IIB-9

Cancer Claims Included in the Industrial Disease Survey[a] by Exposure[b] and Last Claim Status[c]

Exposure[b]	Allowed	Rejected	Claimant Did Not Reply[c]	No Action[d]	Total
Asbestos	12	13	2	1	28
Radiation	3	5	0	7	15
P.A.H.s	4	4	1	0	9
Nickel	13	12	1	1	27
Arsenic	0	1	0	0	1
Other	0	19	1	5	26
Total	**32**	**54**	**5**	**14**	**105**

a See Table IIB-5, note "a."

b A more detailed categorization of exposure substances was established and has been re-assembled into these larger groups for simplification. For example, "radiation" includes not only uranium mining, which is governed by guidelines, but also exposure to gamma radiation e.g. x-rays. This is explained fully in Section 3.1 of this chapter. "P.A.H.s" refer to polycyclic aromatic hydrocarbons and includes coke oven emissions, coal tar exposure, foundry exposures, etc. as explained in Section 3.2.

c Claimant failed to reply to request for evidence and claim was dropped.

d These consist of claims that were set up by the claimant to document excessive exposure to a known carcinogen. As no disability had yet resulted, the claims have been inactive.

2.4 The Appeal Process

The involvement of various worker advocates did not seem to have a significant effect on the rejection rate. Of the 46 claims included in the survey, in which there was participation by a union, federal member of parliament, M.P.P. and/or injured workers consultant, 23 were rejected. In comparison, 32 claims were rejected of the 59 in which no participation of the above groups occurred, as shown in Table IIB-10. Various hypotheses may be invoked to explain these data, and no firm conclusions can be drawn. The lack of an overt pattern suggests that the involvement of pressure groups, which as shown here, is considerable, (and as shown in Section 3) is usually associated with a great deal of supportive research and documentation regarding the claim, does not seem to influence the outcome. (However, as shown in Table IIB-11, those claims initiated by the employer or a health professional had a slightly better outcome than those claims filed by the claimant or by a union. It is likely that 10 of the 17 allowed claims in the "no outside group" category were those filed by the employer. If one subtracts the employer filed claims from the "no outside group" claims in proportion to claim status, as listed in the first row of Table IIB-11a, the rejection rate for the "no outside group" claims is almost 80%, compared to 64% for cases in which "outside groups participated.)

More importantly, whereas the Claims Review Branch reverses about 8% of adverse decisions and recommends alternate action in 36.2% of claims overall (Table IIB-12a), our review of cancer claims did not reveal a single reversal of an adverse decision by that branch (Table IIB-12b). Even more astounding, whereas the appeal process results in the reversal of almost 50% of the adverse decisions brought to appeal (Table IIB-13a), not a single reversal of an adverse decison on a cancer claim was encountered in our review (Table IIB-13b). Interestingly, employers were found to attend about the same percentage of cancer claim appeals as appeals in other types of claims (about half), hence the presence of the employer cannot be a major factor in these rejections. The case studies of adjudication of cancer claims as described below further address the reasons for claim rejection.

2.5 *Under-Reporting of Occupational Cancer*

A look at the statistics quickly reveals that only a very small percentage of cancer cases ever reach the Compensation Board. Only 33 (see Table IIB-2b) of the over 14,000 fatal cancer cases in Ontario in 1979 were recognized as work-related, representing less than one-quarter of one percent. This is significantly less than even the most conservative estimate of the prevalence of occupational cancer (at least 40 times this figure - see Part A of this chapter).

Table IIB-14 lists the number of cancer deaths in Ontario by cancer site along with the number of occupationally related cancers expected from the Doll and Peto[7] estimates. Table IIB-15 lists the total cancer deaths by cancer site in Ontario, along with the number of claims reported and allowed by the Workers' Compensation Board of Ontario as a percentage of the totals in Table IIB-14. Using this approach, only 3.3% of the estimated number of occupational lung cancer deaths in Ontario are currently being compensated. (Using figures from Table IIB-4b i.e. fatal cancer claims allowed, this number would be only 1.6% of the estimated occupational lung cancer in Ontario.) The data indicate that only 13.5% are even reported. Although the Board's medical staff do indeed believe that the cancer compensated represents the true extent of occupational cancer in Ontario, the calculations here strongly suggest that significant under-reporting of occupational cancer is a reality. (It may be argued that the Doll and Peto estimates are not transferable to Ontario. While this may be true, there is reason to believe that the bias in so doing would err on the side of conservatism; Ontario's mining industry, for example, is the source of considerable cancer risk that does not exist in other jurisdictions.)

Peter Barth also looked at the question of possible under-reporting of cancer claims, particularly in relation to asbestos-related cancers.[8] By referring to Selikoff's 10-year mortality study of all members of the International Association of Heat and Frost Insulators and Asbestos Workers in the United States and Canada[9], and setting the 175 fatalities found due to mesothelioma at 100, he indexed the number of fatalities due to lung cancer at 278, laryngeal cancer at 6, and gastrointestinal cancer at 57. He then created a similar index by setting the 53

Table IIB-10

Cancer Claims in the Industrial Disease Survey[a] by Outside Group Involvement and First Claim Status

Outside Group Involvement, Union, MP or MPP, Injured Worker's Consultants, Other)	Allowed Claims						
	Medical Aid Only	Compensation and Medical Aid	Death Benefits	Claim Rejected	Claimant Did Not Reply	No Action	Total
Union Only	-	7	4	17	3	7	38
Union & MP or MPP	-	-	-	-	-	-	-
Union & Injured Workers' Consultants	-	-	-	-	-	-	-
Union & Other	-	-	-	-	-	-	-
MP or MPP Only	-	1	1	4	-	-	6
MP or MPP and Other	-	-	-	-	-	-	-
Injured Workers' Consultants	-	-	-	-	-	-	-
Other	-	-	-	2	-	-	2
Sub-total	-	8	5	23	3	7	46
No Outside Group Involved	3	6	8	32	3	7	59
Total	3	14	13	55	6	14	105

Notes

a See Table IIB-5, Note "a."

Table IIB-11a

Cancer Claims in the Industrial Disease Survey[a] by Person/Group who Filed Claim, and Claim Status

Person/Group Who Filed Claim	Allowed Medical Aid Only Claims	Allowed Compensation and Medical Aid Claims	Allowed Death Benefit Claims	Rejected Claims	Claimant Did Not Reply Claims	No Action Claims	Total Claims
Employer	2	4	4	5	1	6	22
Claimant	-	-	1	8	-	-	9
Union	-	3	1	9	-	6	19
Family Doctor	-	4	2	11	2	-	19
Company Doctor	-	-	1	1	-	-	2
Government Doctor	-	-	-	1	-	-	1
Other[b]	1	3	4	20	3	2	33
Total	3	14	13	55	6	14	105

Notes

a See Table IIB-5, Note "a."

b Represents claims where no single Person/Group could be identified (24.2% of the "Other" claims were allowed.)

Table IIB-11b

Outcome of Claims Initiated by Employers or Health Professionals Compared to Those Initiated by Claimant or Union

	Allowed		Other		Total	
	Claims	%	Claims	%	Claims	%
Health Professionals and Employers	17	38.6%	27	61.4%	44	100.0%
Claimant & Unions	5	17.8%	23	82.2%	28	100.0%
Total	22	30.6%	50	69.4%	72	100.0%

$X^2 = 4.37$, 1; P = 0.037 significant

Table IIB-12a

Claims Review Branch Adverse Decisions and Objections
Total Claims Reviewed, 1979, 1980[a]

	1979		1980	
Recommended Decision				
Reversed	1,573	(8.0%)	1,649	(7.6%)
Confirmed	10,979	(55.8%)	11,455	(53.1%)
Returned for Alternate Action	7,112	(36.2%)	8,477	(39.3%)
Total Referred[b]	**19,664**	**100.0%**	**21,581**	**100.0%**

Notes

a. Source: 1980 Ontario Workers' Compensation Board Annual Report, Table 4, pg. 22.

b. Note that the total number of claims reported in 1980 and 1979 was 444,674 and 460,972 respectively. Of these, allowed compensation claims were 165,221 (37.1%) and 165,454 (35.9%) respectively, and rejected claims were 28,332 (6.4%) and 35,562 (7.7%) respectively. Thus the number of claims that were referred to Claims Review represented 4.4% and 4.7% of total claims respectively, and 69% and 60.6% of rejected claims for each of the two years.

Table IIB-12b

Claims Review Branch Adverse Decisions and Objections
Cancer Claims in Sample Survey

	Cancer Claims Initially Rejected		Cancer Claims Other Status	
Recommended Decision				
Reverse	0	0.0%	0	
Confirmed	54	100.0%	1	(100.0%)
Other	0	0.0%		
Total Referred	54[a]	100.0%	1	
Total Not Referred to Claims Review Branch	1		49	
Grand Total	**55**		**50**	**105**

Notes

a. Note that almost all of the rejected cancer claims were referred to the Claims Review Branch.

Table IIB-13a

Appealed Claims by Last Outcome - Total Claims 1980

	Appeal Heard in 1980	% Allowed	% Undecided	% Unchanged
Appeals Adjudicator	2,79153.0%	8.4%	38.6%	
Appeal Board	1,06441.0%	18.7%	59.7%	
Total	**3,85549.7%**		**50.3%**	

Note

Source: 1980 Ontario Workers' Compensation Board Annual Report

Table IIB-13b

Appealed Cancer Claims by Last Outcome - Sample Survey

	Rejected Claim Total	Allowed	Other	No Change
Appeals Adjudicator:	9	0.0%	4	5
No Hearing	(4)[a]	(0.0)	(3)	(1)
With Hearing	(5)	(0.0)	(1)	(4)
Appeal Board:	5	0.0	2	3
Ajudicator Plus Board	(1)	(0.0)	(1)	(0)
Appeal Board only	(4)	(0.0)	(1)	(3)
Total	**14**	**0.0%**		**100.0%**
Initially Rejected Claims That Did Go To Appeal	41			
Total Rejected Cancer Claims In Sample	**55**			

Notes

Source: See Table IIB-5.

[a] parentheses indicate subsections.

Table IIB-14

Cancers in Ontario that Definitely can be Produced by Occupational Hazards[a]

Cancer Site	Total Deaths With Cancer In Ontario (1979)[c]	Estimate of Percent Work-Related[d]	Estimated Total Occupational Cancer In Ontario By Site
Lung	3397	12.5%	425
Larynx	141	2%	3
Nasal Sinus & Remaining Respiratory Sites (incl. pleura)	55[e]	18%	10
Mesentery, Peritoneum & Unspecified G.I.	1536[f]	10%	153
Bone	51	3%	2
Skin Other Than Melanoma	67	10%	7
Prostate	1000	1%	10
Bladder	461	8%	37
Leukemia	694	8%	56
Totals[g]			

Notes

a. This table is adapted from Doll, R. and Peto, R. JNCI Vol. 66 No. 6 June 1981, p. 1244, table 19.

b. Cancer sites are listed as by Doll and Peto (with the exception of liver cancer which was incorporated into 'unspecified G.I.' to facilitate comparison with Ontario cancer data).

c. From Spengler, R.F. and Clarke, E.A., Review of Cancer Incidence, Mortality Treatment and Survival in Ontario in Cancer in Ontario 1980, The Ontario Cancer Treatment and Research Foundation, Table V.

d. The estimates listed are those derived from Table 19 of Doll and Peto 1981 by proportionately combining the male and female estimates given. (This was done to facilitate comparison with Workers' Compensation Board data which are not listed by sex.) Based on these site-specific estimates, Doll and Peto conclude that probably 4% of all cancers are work-related. The details of the assumptions made in deriving these estimates were summarized in Part A of this chapter. (The estimate for bladder cancer is considered the most reliable of the figures). Although these estimates are considered to be low by many, as discussed in Part A, no site-specific estimates are provided by those who postulate higher estimates; the figures in this table must again be considered "conservative" in light of the debate.

e. This number represents 22 sinus cancers, including "nose, middle ear and accessory sinuses" as well as 33 "other respiratory cancers" as listed in Table VI of the Ontario Cancer Treatment and Research Foundation report (see "c"). As no category for "pleura" was given, it is assumed to have been included in "other respiratory cancers".

f. In the listings used by Doll and Peto "mesentery, peritoneum (including other digestive sites)" was listed as "definitely occupational", whereas no separate estimate was provided for any specific digestive cancers. (Doll & Peto assumed that intestinal cancer is "non-occupational" and that cancer of the colon, rectum, stomach and esophagus are "possibly occupational" allowing a token 1% for males and 0.5% in females in their over-all estimate.) To conform with this reasoning (albeit questionable) only "unspecified G.I." figures were used and deaths from cancer of the colon, rectum, stomach, esophagus and intestines were excluded completely. (The number given in col. 3 is thus actually even lower than it should be.)

g. No totals can be provided in this table. Doll and Peto provided totals in their table by including "other and unspecified cancers" which they felt belonged in the "definitely occupational" category. Insufficient details are provided regarding this category and hence no corresponding Ontario figures can be applied. Moreover, Doll and Peto add 1% of all male and 0.5% of all female cancer of the mouth, esophagus, stomach, colon and rectum, pancreas, connective tissue, kidney, brain, Hodgkin's disease, and non-Hodgkins lymphoma as possibly work-related. Using Ontario data this would add another 35 estimated occupational cancer cases to the total, for an estimate of 738 occupational cancers in Ontario for that year. Doll and Peto's overall estimate of occupational cancers is given as 4% of the total, amounting to 570 cancers in Ontario - slightly lower than the figure derived from applying their methodology to Ontario data.)

Table IIB-15
Cancers Reported to the WCBO as Percentage of Total Estimated Occupational Cancers in Ontario, by Cancer Site

Cancer Site	No. Reported to WCBO[a]	No. Reported To WCB as % of Ont. Cancers*	No. of Estimated Occupational Cancers	No. Reported to WCB as % of Estimated Occupational Cancers**	No. Allowed as % of Estimated***
Lung	68	2.0	425	16.0	(3.3)[g]
Larynx	2 [b]	1.4	3	66.6	
Sinus and other respiratory	14	25.0	10	140.0[e]	(60)
G.I.	7 [c]	0.4	153	4.5	
Bladder	1	0.2	37	2.7	
Skin	2 [d]	3.0	7	28.5	
Leukemia	1	0.1	56	1.8	
Prostate	0	0.0	10	0.0	
Bone	0	0.0	2	0.0	
	95		**703**	**13.5%**[f]	

* Col. 2 Table IIB-14

** Col. 2 as % of Col. 4

*** Col. 3 Table IIB-4A as % of Col. 4

Notes

a. Figures from Table IIB-4a were used as in 60% of the claims in Table IIB-4b the cancer site was not recorded.

b. This would be the upper limit of laryngeal cancer reported to the WCBO in 1980. The category "throat" in the WCB reported statistics may include pharyngeal and tonsillar cancer.

c. This is the total number of G.I. cancer reported to the Board in 1980, therefore it also represents the upper limit of its corresponding category in Table IIB-5.

d. As above; may include melanomas.

e. It is noteworthy that 140% of the "estimated" occupational cancers in this category were reported to the Board. This may either indicate that Doll and Peto's estimate of occupational cancer is too low or that more cancers than are indeed occupational are being reported. As only 6 claims were allowed (Table IIB-4) i.e., only 60% of the estimated number of occupationally-related "sinus and other respiratory" cancers were actually recognized.

f. The number of cancer cases reported to the WCBO represents less than 13.5% of the estimated occupational cancers in Ontario. (For the reasons listed in the notes to the previous table, this is a conservative estimate.)

g. Only 3.2% of the estimated number of occupational lung cancers were compensated in 1980. If the figures in Table IIB-4b were used instead, only 1.6% of the estimated number of occupational lung cancers would be compensated. (The latter, representing fatal cancer claims, is probably more appropriate.)

mesothelioma claims allowed and rejected to an index of 100, as shown in Table IIB-16. He showed a small excess in the number of claims for laryngeal cancer and a substantial shortfall of claims for the other two groups, especially lung cancer. Barth also included a comparison based on allowed claims only, as also shown in Table IIB-16. He concluded that the disparity between the Selikoff data and the Workers' Compensation Board's data suggested that a substantial number of potential cancer claims are not being reported to the Board, with the extent of the disparity being dependent on the number of mesotheliomas being missed. Barth stressed that these results are only suggestive and should be judged with caution. First, he pointed out, that Selikoff's population is very different from that seen by the Workers' Compensation Board with differences in duration and intensity of exposure; secondly, Workers' Compensation Board claims involve living persons whereas Selikoff's data are from fatalities only; thirdly, Selikoff's data regarding cause of death are much more precise. (In fact, less than half the mesothelioma cases were actually recorded as such on death certificates.) Nonetheless, Barth did note that the data suggest that the Workers' Compensation Board is not receiving claims for asbestos-related cancers, particularly lung cancer, and that additional inquiry and further outreach activity might be appropriate. (Unfortunately, the data necessary to provide a much more accurate estimate do not exist. This, in itself, attests to the need for better data collection, as will be repeatedly alluded to below.)

Although the rejection rate and the procedures whereby these rejections are routinely confirmed are certainly problematic, it can still be concluded that under-reporting of occupational cancer seems to overshadow these issues as a reason for undercompensation of occupational cancer.

2.6 *The Delay Factor*

In presentations to the Weiler Inquiry, some people felt that the difficulties in getting claims allowed, "often humiliating investigations", and long cumbersome delays discourage many from filing claims. As shown in Table IIB-17, claims usually take only a few days to process and even the more serious industrial accident claims take an average of about four weeks from claim set up to the date a cheque is mailed to the claimant. Even those requiring secondary input take an average of only 7 1/2 weeks. In contrast, cancer claims take an average of over 25 weeks to adjudicate (see Table IIB-18a) and another 3-4 weeks for the claimant (or survivor) to receive benefits or written notification (see Table IIB-18b). Our study also found that cancer claims that are appealed may spend over a year in the appeal process as discussed in the next chapter. Thus the delay factor is not negligible.

2.7 *Scientific Evidence and Medical Knowledge*

It has been said that doctors are unwilling to file claims if supporting scientific evidence is scarce or uncertainty remains. In the review of 105 cancer claims carried out for this study, the attending physician was felt to be the initiator of only 19 of the cancer claims or 6% of those in which the "initiator" could be determined (see Table IIB-11a, last column). However, the attending physician participated in over 70% of the cancer claims filed. It is difficult to assess the reasons behind the lack of reporting of the other at least 86% of occupational cancers (i.e. the

Table IIB-16

Types of Cancer in Asbestos-Related Claims, Indexed[a,b]

Site	Observed Causes of Death, Cancer in Asbestos Workers (Selikoff)	All Claims to the WCB, Cancer, Asbestos Involvement	Accepted Claims by the WCB, Cancer, Asbestos Involvement
Mesothelioma	100	100	100
Lung	278	121	110
Larynx	6	25	8
Stomach, Colon-Rectum, Esophagus	57	26	15

Notes

a Indexed so that mesothelioma = 100

b Prepared by Peter Barth for the Royal Commission on Asbestos

Table IIB-17

Claims Processing Times[a]

Claim Type[b]		Mean No. of Work Days[c]	% of Total Claims Surveyed
B1		2.5	53.0%
B2		19.8	9.7%
C1		2.4	2.2%
C2	- no secondary input	19.2 (approx. 4 weeks)	28.1%
	- secondary input	38.4 (approx. 7 1/2 weeks)	

Notes

a Source: Initial Payment Performance Survey, May 1981.
Program Planning and Statistical Services, WCBO.

b B1 Form 7 (Employer's Report) is the first piece of mail received.
B2 Form 8 (Doctor's Report) is the first piece of mail received.
C1 Similar to B, except there is a possibility of extended disability in the future.
C2 Extended disability claims.

c Calculated from date first document received at the WCB to date cheque is mailed.

Table IIB-18a

Frequency Distribution of Cancer Claims in the Industrial Disease Survey[a] by Weeks from Date Claim Set Up to Date Initial Decision Reached, and First Claim Status

Calendar Weeks to Initial Decision	Allowed Medical Aid Only Claims	Allowed Compensation and Medical Aid Claims	Allowed Death Benefit Claims	Rejected Claims	Claimant Did Not Reply Claims	No Action Claims	Total Cum. Claims	%
0	-	1	-	-	-	-	1	0.9
0 to 1	-	-	-	-	-	3	3	3.8
1 to 2	-	-	-	-	-	-	-	3.8
2 to 3	-	-	-	-	-	1	1	4.7
3 to 4	-	-	-	-	-	-	-	4.7
4 to 5	-	-	-	1	-	-	1	5.6
5 to 6	-	-	-	-	-	-	-	5.6
6 to 7	-	2	-	-	-	2	4	9.4
7 to 8	-	1	-	2	-	-	3	12.3
8 to 9	-	1	-	2	1	-	4	16.1
9 to 10	-	3	-	1	1	6	11	26.6
10 to 11	-	2	-	2	-	-	4	30.4
11 to 12	1	-	-	2	-	-	3	33.3
12 to 13	-	-	-	1	-	1	2	35.2
13 to 26	1	1	6	21	2	1	32	65.7
26 to 52	-	2	4	15	2	-	23	87.6
52 to 104	-	-	3	8	-	-	11	98.1
104 to 156	1	-	-	-	-	-	1	99.0
Unknown	-	1	-	-	-	-	1	100.0
Total	3	14	13	55	6	14	105	
Mean Weeks	50.7	12.5	34.1	29.8	24.7	7.2		28.7[b]
S.E.M.	35.2	2.8	5.1	2.8	6.1	1.2		2.5
Standard Deviation	60.9	10.1	18.5	20.8	15.0	4.6		22.5

Notes

a See Table IIB-5, Note "a."
b Calculated excluding "no action" claims.

Table IIB-18b

Frequency Distribution of Cancer Claims in the Industrial Disease Survey by Weeks from Date Initial Decision Reached, to Date Initial Decision Conveyed to Claimant, and First Claim Status

Calendar Weeks to Notify Claimant	Allowed Medical Aid Only Claims	Allowed Compensation and Medical Aid Claims	Allowed Death Benefit Claims	Rejected Claims	Claimant Did Not Reply Claims	No Action Claims	Total Cum. Claims	%
0	-	1	2	6	3	13	25	23.8
0 to 1	-	4	3	40	1	-	48	69.5
1 to 2	-	1	2	5	-	-	8	77.1
2 to 3	-	-	3	-	1	-	4	80.9
3 to 4	-	1	2	2	-	-	5	85.7
4 to 5	-	-	-	1	-	-	1	86.6
5 to 6	-	1	-	1	-	-	2	88.5
6 to 7	-	-	1	-	-	-	1	89.4
7 to 8	1	1	-	-	-	-	2	91.3
8 to 9	-	-	-	-	-	-	-	91.3
9 to 10	-	-	-	-	-	-	-	91.3
10 to 11	-	-	-	-	-	-	-	91.3
11 to 12	-	-	-	-	-	-	-	91.3
12 to 13	1	-	-	-	-	-	1	92.2
13 to 26	-	3	-	-	-	-	3	95.1
26 to 52	-	-	-	-	1	-	1	96.1
52 to 104	-	-	-	-	-	-	-	96.1
104 to 156	1	-	-	-	-	-	1	97.1
Unknown	-	2	-	-	-	1	3	100.0
Total	3	14	13	55	6	14	105	
Mean Weeks	50.8	5.8	1.9	1.7		0.0		3.4
S.E.M.	40.8	2.1	0.6	0.8		0.0		1.4
Standard Deviation	70.7	7.3	2.0	6.4		0.0		14.2

fraction of occupational cancers estimated to go unreported). Many anecdotes have been conveyed to this Inquiry as to the reluctance of physicians to file claims, and hypotheses have been put forward regarding reasons for this, most of which revolve around the policy of the Board with respect to burden of proof. The general lack of training in the field of occupational medicine has also been repeatedly mentioned.

2.8 Outreach Programs

The fact that under-reporting of occupational cancer exists, attests to limitations in outreach programs. In an attempt to bring worthy claims to the Board, various outreach programs were indeed begun by the Ontario Workers' Compensation Board. One such program was the asbestos outreach which began in 1976. WCB personnel visited or called numerous plants, the Ontario Federation of Labour, and key labour unions to inform them of board policy regarding conditions of eligibility for compensation of workers who had developed possible asbestos-related diseases. The program specifically focused on publicizing the new policy for compensating gastrointestinal cancers and later any asbestos-induced cancer, however, Barth points out that much less attention was given to publicizing the availability of compensation for laryngeal cancer in asbestos workers. Also, he points out, that only the industries most obviously at risk were included. By July 1980 only 22 claims had been established directly as a result of this program, of which 19 were denied, 2 were allowed and 1 was withdrawn. Barth does mention that the outreach program may have had indirect benefits not indicated by these statistics.[10]

Other efforts of the Ontario Workers' Compensation Board to inform the community about occupational cancer and other industrial diseases, include various seminars, articles and letters directed at the medical community. Regardless of the evaluation of the success or failure of these endeavours, it should be noted that outreach programs of any kind are very uncommon among worker's compensation agencies, (although often other government agencies are entrusted with this role). Thus again, while much more could be done, the Ontario WCB does compare favourably to other jurisdictions.

As was shown in Chapter I, a great many more cancer claims have been allowed in Ontario than in all the other provinces combined. Given the much larger industrial base in this province, and the fact that more than 40% of all Workers' Compensation Board claims in Canada are handled in Ontario, it is not surprising that both the policy development and the quantitative experience with occupational cancer are much greater in Ontario than in other Canadian jurisdictions.

Notwithstanding Ontario's seemingly superior record by other jurisdictional standards, many victims of occupational cancer, if not the great majority, are not being compensated. Suggestions to the Weiler Inquiry for possible methods of improving the provision of justice to victims of occupational cancer have been varied. Some suggest that the guidelines themselves act as a hindrance and should be eliminated, with each claim adjudicated on its own merit. Some insist that adjudication should be more strongly based on awarding the benefit of doubt to the worker, (which, in fact, is the stated policy of the Board). What these proponents mean, however, is that the onus should be on the employer to prove lack of work-relatedness and in the absence of scientific evidence, the claim should be considered compensable. Others obviously object to this suggestion. Some have recommended that once a clear relationship is established between a work process or chemical and a cancer, it should be placed in a schedule of diseases with minimally restrictive criteria, providing a statutory presumption in favour of granting compensation in cases when these criteria are met. Still others feel that emphasis should be placed on the guidelines themselves with their numbers greatly expanded, rather than including cancers in a schedule where presumption favours the worker. Possible cost-sharing methods between employers and society as a whole have also been discussed and will be addressed elsewhere.

The relative merit of each of these suggestions can best be judged in appreciation of the scientific background and the current process of development of guidelines for the adjudication of the various cancer claims. These will be discussed, followed by case presentations illustrating current policy.

3. The Decision-Making Process in the Adjudication of Cancer Claims

3.1 Radiation and Cancer

Issues of contention in the adjudication of cancer claims in workers exposed to radiation basically revolve around, first the degree of exposure in individual workers, (in terms of length of exposure, intensity of exposure, and sufficient documentation thereof) and secondly, the probability that that particular level or type of exposure gave rise to or contributed to the cancer that ensued. As well, the issues of sufficient latency period, out-of-province exposure, and tumour site and cell type have emerged as contentious, as perhaps will the question of medical exposures to radiation. In order to appreciate the controversy with respect to the decision-making process in the adjudication of these claims it is necessary to have some understanding of the state of scientific knowledge in this field. A statistical overview of the Board's experience with claims for radiation exposure and the evolution of the Board's policy on these matters will then be presented, followed by some illustrative case examples of the difficulties identified.

3.1.1 The scientific background

(a) Radiation and its effects: Ionizing radiation, i.e. radiant energy which either directly or indirectly alters chemical bonding by dislodging electrons from molecules in the material through which it passes, includes emissions produced by radioactive materials, fissioning atoms, x-ray machines and particle accelerators, and excludes other ("non-ionizing") radiation such as infra-red, ultra-violet, radio waves, etc.

The effects upon living tissue of exposure to ionizing radiation may be classified into two broad groups:

(a) **Genetic:** Those changes within the reproductive cells which do not affect the person exposed, but which show up in subsequent generations; and

(b) **Somatic:** Those effects which show themselves in the person exposed, but which are not passed on to subsequent generations. The latter can be divided into early effects, e.g. radiation burns, loss of hair, acute radiation syndrome, etc. and late effects, e.g. leukemia, anemia, cancer, cataracts, etc.

Ionizing radiation can be divided into alpha, beta, gamma, x-ray and neutron radiation. Biological effects are basically similar for the different types of ionizing radiation, but they vary in their ability to produce biological changes, with alpha most effective and gamma less so. Factors of tissue penetration are important. These are low for alpha and variable for x-rays depending on frequency. This makes for considerable difficulty in estimating actual internal tissue dosages to any given organ - particularly from mixed sources. Alpha radiation travels a short distance, is densely ionizing, and hence, is the greater danger internally (for example, uranium miners exposed to radon gas and daughters which may be inhaled, have a clearly higher incidence of lung cancer, but no documented increase in leukemia). Beta radiation presents a slight external hazard, e.g. skin burns, cataracts, as well as an internal hazard if it gets in. Gamma radiation, either emitted along with alpha and beta particles, or as electromagnetic radiation in x-rays, is largely an "external hazard" penetrating extensively and therefore capable of damaging many different organ systems.

Radiation is measured by the amount of ionization it produces. The basic unit of radiation, the roentgen, named after William Conrad Roentgen, discoverer of x-rays, is used in the measurement of x-ray exposure. "Rad" refers to the "radiation absorbed dose", and the REM, "rad equivalent man" refers to the dose of radiation which produces a biological effect equivalent to the absorption of one rad of x-radiation.

Uranium miners are primarily exposed to radioactivity in the form of radon gas which is formed as part of the decay series of uranium and radium. The miners inhale the shortlived decay products of radon, known as radon daughters which bombard the lung tissue increasing its likelihood of malignant transformation. A special unit for measuring radon gas and radon daughters has been developed called a "working level". The working level (WL) measures the ionizing power of the radon daughters. A uranium miner exposed to one WL for 170 hours, the average number of hours worked per month, is said to have worked one working level month (WLM).

There is considerable divergence of opinion as to the correction factors for relative biological effectiveness that should be applied (e.g. the equivalent whole body radiation to lung tissue compared to one WLM of alpha particle exposure is often considered as 5 REM, but opinions seem to range from 1 to 50 REM.)[11]

Personal monitoring of whole body exposure to gamma emissions and x-rays is reasonably accurate. In Canada, film badges are provided for this purpose which are all processed and recorded in Ottawa, except for the Chalk River workers. However, good monitoring is not available for personal exposure to most alpha emissions, and the presently available film badges for gamma radiation are apparently not adequately sturdy for use by miners either. (Also, until very recently, monitoring for tritium was completely non-existent, although it is now known to significantly contribute to the total radiation exposure in nuclear reactor workers[12].)

Exposure to x-rays has been known for years to be associated with adverse effects such as skin cancer and leukemia. Both animal and human data indicate that radiation can produce cancer in a wide variety of organs. Although there is no "safe" level, a permissible dose of five REM has been set. There is considerable controversy in this area, however, as studies suggest that even levels below five REM may be associated with increased damage to chromosomes.[13]

Epidemiological data in human cancer after radiation exposure come largely from studies of the populations at Hiroshima and Nagasaki, studies of groups of patients with past history of exposure to therapeutic or diagnostic x-rays, and the hotly debated follow-up of workers at the Hanford Nuclear Facility in Washington State.

Dose estimates in the atomic bomb survival studies are complicated and inaccurate.[14] Attempts to revise these estimates have recently been attempted[15]. Moreover, this event produced a single major radiation dose, which may not be applicable to the occupational setting. Some authors have suggested that the tumorigenic response is greatly increased by fractionating a dose,[16] whereas others suggest just the opposite.[17] The follow-up study of patients who received therapeutic and diagnostic x-ray exposure confirms a causal relationship between a wide variety of cancers and radiation exposure, but gives only a rough indication of the dose-response.

The data from the Hanford Nuclear Facility are as important as they are controversial. The experience of a work force with dose levels of exposures comparable to those commonly found in Ontario was examined. Mancuso et al. examined the records of workers who died within 49 years of Hanford going into full production and found that men who eventually developed fatal cancer had been more often and more intensively exposed to external radiation than men with other causes of death. Specifically, increased risks were found for bone marrow cancer, reticuloendothelial system neoplasms (including lymphoma, lymphosarcoma, Hodgkins disease, etc.), pancreatic cancer and to a lesser extent lung cancer.[18] (Interestingly, no increased risk for leukemia as previously demonstrated was noted.) Controversy arose because the estimated increase in risk (per unit dose) at relatively low dose levels (less than 30 rads) was approximately 10 - 20 times greater than previous studies had shown.

(Mancuso et al. used the methodology of proportionate mortality analysis as well as relating the proportion of cancer to cumulative dose. Criticisms of the methodology included the fact that the SMR's were calculated without attempting to standardize to the age-distribution of the Hanford deaths, the utilization of both the 7th ICD revision (for expected deaths) and the 8th revision (for observed deaths) created differences, the denominator in the EMR calculation (excess mortality based on radiation doubling dose relative to a standard risk) was all causes, rather than all cancers, which, due to the "healthy worker effect" created erroneous results, and the "expected deaths" were taken from a single year (1960) which was considered erroneous as cancer frequency had changed significantly (e.g. for lung cancer) over the 29 years of the study.[19])

Various attempts to rework these data have been published.[20] The opinions of experts vary from considering that no valid conclusion at all could be reached, to an acceptance that the data show an increase in cancer of the pancreas and multiple myeloma and a suggested increase in a number of other types of cancer. A second study conducted by Mancuso's team extending the original follow-up period, confirmed the previous findings.[21] Yet a third study they conducted using a different methodological technique also allowed them to reiterate their conclusions, this time indicating a particular group of radiosensitive cancers.[22]

The varieties of cancer definitely (++) or probably (+) associated with radiation as determined by BEIR (the Advisory Committee report on Biological Effects of Ionizing Radiation), the publication of the National Academy of Sciences, and other sources are shown in Table IIB-19. The International Commission on Radiological Protection (ICRP), BEIR and the United Nations Scientific Committee (UNSCEAR) have developed risk estimates for various types of cancer in terms of the number of extra cases per million persons per year per REM (the absolute risk). Much controversy exists over these estimates especially given the relatively short follow-up periods in the studies on which they are based and other methodological limitations. BEIR developed the concept of the "doubling dose", now often used to mean the dose needed to double the relative risk. Problems exist with this concept because the "natural risk" for different tumours varies so widely in different studies of different population groups. Swartz and Epstein have criticized these risk estimates for having been derived from the A-bomb survivors, and from studies of patients with spondylosis who tend to die prematurely from this debilitating disease, perhaps before cancer has had a chance to become manifest.[23] In short, there is no consensus as to how risk should be assessed.

Table IIB-19

Cancers Definitely (++) and Probably (+) Caused by Radiation*

Cancer Type	A-Bomb Survivors	Occupational Exposures	Diagnostic/Therapeutic Radiation	
			Adults	Children
Leukemia	++	++	++	++
Female Breast	++		++	
Thyroid	++			++
Lung	++	++	++	
Stomach	++		++	
G.I.	++		++	
Bone		++	++	++
Sinuses		++	+	
Bladder			+	
Brain				+
Cervix	+		+	
Lymphoma	+	+		
Multiple Myeloma	+	+		
Ovary			+	
Salvary Glands	+		+	
Skin		++	++	+
Vagina			+	
Pancreas			+	
Rectum			+	

* From: Chovil, An Internal Report to the Workers' Compensation Board Summarizing the Scientific Background on Cancer and Radiation August 29, 1980.

Dr. Letourneau and Dr. Waight at the Radiation Protection Branch in Ottawa, who advise the Canada Pension Commission on claims, are apparently of the opinion that the only practical way is to make individual assessments of the risk of the radiation cancer of the type involved based on the patient's exposure, and to compare these results with those for the spontaneous development of such a tumour using figures developed from provincial or federal vital statistics.[24] Other options with respect to compensation claims will be discussed in Section 3.1.2

(b) Epidemiology of primary lung cancer in uranium miners: As most cancer claims due to exposure to ionizing radiation have come from the uranium mines, and there has been a fair amount of study on this issue, the epidemiology of lung cancer in uranium mines is instructive to review.

An increased risk of developing lung cancer following exposure to radon and its daughters has been observed in studies in Germany,[25] Czechoslovakia,[26] the United States,[27] Britain,[28] Newfoundland[29] here in Ontario.[30], and most recently in Sweden.[31] The increased risk of fatal lung disease in association with various mining operations has in fact been recognized for more than four centuries.

In the sixteenth century, Paracelsus described the disease of metal miners in the Erz Mountains at Schneeberg, Germany. The miners, noting that many of their colleagues were dying of respiratory ailments, called the disease "mountain sickness". The relation to dust inhalation was documented by Agricola shortly afterwards.[32] In 1879, Harting and Hesse showed that three-quarters of these deaths at Schneeberg were due to lung tumours[33] and it was later shown that mountain sickness was indeed bronchogenic carcinoma.[34] The air in the Schneeberg mines was known to be radioactive and this is felt to have been the most likely cause of the cancer hazard.

In 1926, the diagnosis of lung cancer was made in uranium miners at Joachimsthal, Czechoslovakia,[35] and of 53 active or retired miners who died in 1929-1930 and 1933-1937, 24 (45%) were found to have died from lung cancer.

As early as 1949, the year uranium was discovered in Elliot Lake, the Colorado Department of Health suspected that they were seeing an increased incidence of lung cancer in their uranium mines, but resisted attempts to study the situation. As Radford noted,[36] when Heuper attempted to establish a register in 1948 for purposes of conducting an epidemiological study of the lung cancer hazard to Colorado uranium miners and millers, the authorities claimed that "scientists recommending such a fact-finding epidemiological procedure in Colorado were displaying 'bad scientific judgment' and deserved to be dismissed from the government service. They insisted, moreover, that any mention of the extensive evidence recorded since 1879 concerning occupational lung cancer hazards in Saxon and Bohemian radioactive mines in Schneeberg and Joachimsthal be suppressed at medical conferences as not being in the 'public interest'."[37]

By the early 1950's, the Workers' Compensation Board of Ontario had expressed concern about potential industrial disease from uranium mining: "Apart from the silica hazard, the Department of Mines is not completely assured that there is not a further hazard from uranium. They do not think that there should be any concern during the early development work, although they are keeping in close touch with the operations."[38] The Department of Mines communicated closely with the Atomic Energy Control Board (AECB) in Ottawa and with Washington regarding the American experience.

By 1957, at the time that Elliot Lake was gearing up for its boom years, the U.S. Public Health Service predicted an imminent lung cancer crisis for the Colorado miners unless radiation levels in the mines were reduced.[39] In 1964,[40] and later in 1969,[41] Archer and Associates published the results of their study showing that indeed there was a statistically significant excess of respiratory cancer deaths in the Colorado mines in each exposure category down to and including 120 to 359 WLM, inexplicable by smoking habits or place of residence. An excess of small-cell undifferentiated or oat-cell lung cancer was specifically noted, increasing in frequency with increasing exposure. The radon daughter concentration in the U.S. uranium mines was thought to be high in the early post-war years and probably comparable to pre-World War II concentrations in Joachimsthal and Schneeberg mines.[42]

By the late 1960's, the fluorspar miners in Newfoundland were found to constitute another mining population exposed to an increased lung cancer risk[43]. A detailed investigation of the health of the miners spanning the time from 1949 to 1971 (excluding the first ten years from start of mining) showed that fluorspar mines in this region had a death rate from lung cancer 29 times that anticipated from study of the unexposed population. The difference was again shown to be unrelated to cigarette smoking or pneumoconiosis and investigation showed that the mine air was radioactive to a degree comparable to that in uranium mines.[44] No radioactive ore was present in the mines and activity was shown to emanate from radon daughters dissolved in water seepage into the mines. Following this study the Workers' Compensation Act of Newfoundland was amended to allow cancer claims in "individuals who at any time worked underground in the fluorspar mines". The Royal Commission investigating this matter wrote:

> While there are insufficient statistics to prove that these (lung cancer as well as cancer from the nose and mouth to the trachea) might have been caused by radiation, it cannot with present knowledge be disproved and indeed there is some scientific evidence to suggest that radiation is the causative factor. Such cases should receive compensation.[45]

The hematite (iron ore) miners in Britain were also found to have a lung cancer risk 1.7 times that of comparable local groups or national experience.[46] Here again, the source of radon in these mines was thought to be seepage of water and the mean radon concentration was estimated to be about 1 WL,[47] adding to the evidence that there is indeed a slight increase in risk of developing lung cancer after several years of exposure to a mere several working levels.

Meanwhile uranium mining had begun in two areas in the Province of Ontario, Elliot Lake and Bancroft, in 1954. It grew rapidly with more than 15 mines operating and over

11,000 persons employed at the peak. Bancroft ceased production completely in 1964 and by 1975, employment had dropped to 1,351 at Elliot Lake. Despite all the evidence of a cancer risk, radiation exposure was considerable at almost all the mines through the peak years and considered to be high in two of the five remaining mines until 1968.[48] (Meanwhile, despite the acknowledgement of considerable radiation exposure, not a single one of the over 100 radiation-related cancer claims received at the WCB by 1968 was accepted by the Board as work-related as discussed in Sections 3.1.2 and 3.1.3 below.)

In 1970, the Ministry of Health of Ontario began a study of deaths in uranium miners. A nominal roll of each hardrock miner with five years' experience underground or one month underground exposure in an Ontario uranium mine was established at the Workers' Compensation Board. Deaths were identified by a computer match of the nominal rolls with the death records of Statistics Canada. The second report of the results was presented in 1974, but never published,[49] and the work was updated and reviewed in 1976 by the Royal Commission on Health and Safety of Workers in Mines.[50] Hewitt showed again that the risk of cancer was related to cumulative radiation exposure and that no threshold was demonstrable.[51] Later it was confirmed that if a threshold existed it could not be greater than 61 WLM and was unlikely to be greater than 10 WLM.[52] Shannon[53] reviewed the data from the Hewitt analysis to again suggest that there is probably no threshold for radiocarcinogenesis. Archer concluded that the existence of a threshold for radiation-induced cancers at any level above 20 or 30 WLM is highly unlikely[54] and has recently reiterated that conclusion.[55] The absence of a threshold is thus consistent with widespread scientific thought.

In 1981, an epidemiologist employed by Ontario's Workers' Compensation Board published a review of lung cancer in uranium miners in Northern Ontario.[56] Twelve cases of lung cancer were observed in the period 1960 to 1964, compared to the expected 4.6. He noted that "as the median latency for this group was five years, it would seem possible that a period as short as this may occur under some conditions". A dose-response effect was acknowledged at all levels, both for squamous and oat-cell tumours, although with an apparent difference in strength, as previously reported in Czechoslovakia.[57] A difference in the proportion of the types of tumours produced by short-term as compared to long-term continuous exposure was suggested. The study reiterated concern with respect to attributability:

> It has been customary (particularly for medical-legal purposes) to think of cancers in some workers as being radiogenic and those in others as being 'spontaneous'. This dichotomy does not equate well with current theories of the causation of cancer by mutation, and the associated processes of

> syncarcinogenesis and tumour promotion. It seems more probable that a whole spectrum of syncarcinogenic responses is being viewed with the radiation contribution ranging from minimal to maximal. This would fit with the apparent demonstration of a dose response at all levels.

The Ontario Miners' Mortality Study begun in 1977 is still underway and may serve to add further insight into some of the more intricate issues.[58]

Meanwhile, a recent study of Swedish iron miners who had been exposed to low levels of radiation, and followed for nearly their lifetime, has shown that smoking and radiation-induced effects are additive, not multiplicative as previously thought. After ten years from beginning mining, 50 deaths from lung cancer were observed, 18 of which were in non-smokers. It had been calculated that the expected rate for non-smokers was 2.0 and that for smokers was 12.6, giving a relative risk of 9 for non-smokers and 2.54 for smokers. (The absolute risk, correcting for amount smoked, came out to be 16.3/(10^6 person-years.WLM) for non-smokers and 22.3 cases of lung cancer/(10^6 person-years.WLM) for smokers.) The latency period was long, and did not seem to be affected by smoking.[59]

(c) "Acceptable risk" and radiation standards Before proceeding to Board policy and possible options in adjudicating compensation claims, it is useful to briefly review the sequence of changes in permissible radiation levels in this province. Although the question of prevention is dealt with at length elsewhere, reference to "permissible levels" is occasionally made in compensation cases and therefore merits comment.

There seems to be general agreement that the dose-response curve shows no threshold and that any level of exposure to radon and its daughters may lead to an increased risk of developing cancer. The level of "acceptable risk" used to set standards in Canada has itself been the source of considerable controversy, both in terms of the generality ("who decides what risks are acceptable") as well as the particularity ("how much exposure constitutes what level of risk"). The ICRP has set recommended standards which have changed from time to time over the years. Initially these standards were set with a view to avoiding excess mutations among the population in the reproductive ages. They were subsequently justified on the basis that they would not produce excess cancers. Most recently it seems that the concept has become that the standard chosen should not permit an increase in the risk of cancer greater than the risk of accidental death which would be incurred by a worker from other industrial hazards over a lifetime in a well-run industry. One may assume that as efforts proceed to decrease the health and safety hazards in industry, the uranium industry will correspondingly alter its "acceptable levels".

The Atomic Energy Control Board was established in 1946 under the Atomic Energy Control Act "to make provision for the control and supervision of the development, application and use of atomic energy."

The standard for radiation exposure in Ontario uranium mines has been influenced by two main sources, the recommendation of the ICRP, and U.S. practice and legislation. Close communication between Ontario and Ottawa is maintained. When mines began production in 1955, Ontario adopted the ICRP recommendation that a maximum permissible concentration of radon and its daughters in air be 100 pci per litre, equivalent to the WL recommendation adopted in U.S. mines. However, enforcement was lax. In 1959, the ICRP revised its recommendation to 30 pci per litre or 3.6 working level months per annum as a maximum for a 40-hour week. Although there was apparently an informal consensus that it was desirable for Ontario to shift to the lower guideline, the higher level, equivalent to 12 WLM per annum continued in Ontario as was the case in U.S. mines and was endorsed in 1967 by the Ontario Mines Engineering Branch. As the excessive risk of lung cancer was becoming clear, the level was reduced to 8 WLM effective January 1st, 1973 and to 6 WLM effective January 1st, 1974. The current level of 4 WLM per annum was made effective as of January 1st, 1975.[60]

In summary, the link between radiation exposure and cancer has been know for centuries. Different forms of radiation have different characteristics that result in varying degrees of likelihood of producing cancer at one site as opposed to another. However the ultimate effect of all forms of radiation on human cells is essentially the same and the issue is one of probabilities. Methods of measurement of radiation exposure, particularly to alpha emissions, leave much to be desired. Both squamous and the more uncommon oat-cell type of lung cancer have been linked with uranium mining, however the latter is more prevalent relative to its existence in the general population. A latency period as short as five years or less has been postulated under some circumstances, but longer than 40 years in others. There seems to be no level of radiation exposure at which the risk of developing cancer is not increased to some extent. There is still much uncertaintly as to what level of exposure constitutes what level of risk, let alone how much "risk" is acceptable.

3.1.2 Workers' Compensation Board Policy on the Adjudication of Cancer Claims Associated with Ionizing Radiation

In 1940, item 9 "any disease due to exposure to x-rays, radium or other radioactive substances" was added to Schedule III and as early as 1955, concern with respect to the adjudication of claims specifically resulting from uranium exposure was expressed. The decision was made at that time that should a claim be received, it was to be considered under item 9 in Schedule III. It was specifically noted that exposure would have to be shown and reference was made to three previous claims for radioactive exposure where proof of exposure had been problematic.

In 1962 the Atomic Energy Committee of the International Association of Industrial Accident Boards and Commission (I.A.I.A.B.C.) conducted a brief survey of the experience of State Workmen's Compensation Agencies in handling cases in which radiation was reported as the cause of injury or disease. The results of the survey indicated that 11 State Workmen's Compensation Agencies processed a total of 88 cases during the preceding three calendar years resulting from x-ray, ionizing radiation or other radiation source, and included effects such as leukemia, bone cancer, dematitis, cataracts, brain damage, tumours, etc.

Another such survey took place in 1963 and the Ontario Workers' Compensation Board's response indicated that during the previous five years 37 claims had been established for radiation injury or disease in this province. Of these, 14 were allowed, 3 were rejected and 20 were pending at the time. (It was noted however, that none of these claims had been for leukemia, bone cancer or other effects with long latency periods.)

By 1968 about 100 claims had been filed for radiation exposure. It was stated at that time that a claim should receive favourable consideration if credible evidence established that: (a) the claimant was exposed to ionizing radiation at work, (b) the disability or disease from which the claimant was suffering was significantly more common among radiation-exposed than among unexposed persons, and (c) the nature and amount of the claimant's radiation dose was sufficient to be a significant factor in causing the disability or disease. No specific acceptance criteria existed for uranium cancer claims and the Board was dealing with them on an ad hoc basis in consultation with a senior medical consultant with the then Department of Health.[61]

The first claim for lung cancer in a uranium miner was accepted in Ontario in 1970.[62] The allowance of this claim was based upon the data from the Colorado observations and the Czechoslovakia data. A probability of cancer being radiation-induced instead of being caused by other factors was calculated from a series of regression equations of mortality estimating the effect of a low cumulative dose of radiation exposure on the frequency of lung cancer at that level. This method received a great deal of criticism and was discontinued after 1972. Thereafter, each case was adjudicated based upon whether or not the tumour was of the oatcell type, the claimant had a cumulative exposure in excess of 120 WLM, the latency period was in excess of ten years, and the claimant had commenced mining prior to 1960 and had worked ten or more years underground.

At the recommendation of the Department of Health one case of oat-cell cancer was allowed in an individual who had accumulated greater than 120 WLM in about four years, despite a latency period of only five years. By the end of 1979, 54 cases of lung cancer attributable to ionizing radiation had been allowed (53 cases of lung cancer in uranium miners and 1 in a worker involved in refinery operations). A profile of these cases, including the age, year of death, lung cancer type, years of exposure, latency and WLM is included in Appendix 2. By August, 1981, this figure had risen to 84 accepted lung cancer claims in uranium miners. The number of cancer claims associated with ionizing radiation that have been allowed as well as rejected over the last few years is show in Table IIB-20 (It should be recalled that claims for cancer other than lung could not be included in a table such as this, as rejected claims are not routinely recorded, for reasons explained in Section 2 above.) At a recent conference on mining hazards held in Colorado, a senior executive of the Workers' Compensation Board's Medical Services Division, pointed out that the 80 to 40 allowed-to-rejected claims in Ontario is consistent with the observed-to-expected ratio and therefore, he felt, is quite appropriate.[63] As well, 8 claims for skin cancer associated with ionizing radiation were allowed (including 4 in physicians.) Also, 2 cases of leukemia had been allowed (1, in an x-ray technician and 1, in a worker associated with the processing of uranium. Just recently, it was announced that 2 cases of cancer were allowed in workers from the Atomic Energy of Canada's Chalk River nuclear research reactor. One died of leukemia, the other is seriously ill with cancer of the lymph glands. A third case is pending.

The Development of Guidelines: In April, 1976, the first guidelines for adjudication of lung cancer in uranium mines were approved. Based on the then current world literature, specifically the Colorado study, a number of presumptions emerged. First, oat-cell carcinoma was considered the common cancer related to radiation effects, although it was pointed out that oat-cell cancer does occur without any history of radiation exposure. Secondly, it was noted that lung cancer is significantly increased in workers who have accumulated 120 WLM or more of radiation exposure. (The rationale for this number was largely that the Czech data had indicated that with 50 WLM, the standardized mortality ratio (SMR) was 1.5, and in the Colorado study 90 WLM produced a SMR of 1.6. Also, in 1975, the AECB had concluded that no significant excess of lung cancer in miners existed with cumulative exposure less than 120 WLM).[64]

It was also appreciated though, that prior to 1968, no individual records were kept regarding the radiation exposure of workers in the Elliot Lake area, however it was felt that favourable consideration should be given to a claim of a worker who had: (1) at least five years' underground exposure prior to 1968 and cancer of the oat-cell type or (2) had more than ten years' exposure underground prior to 1968 for any type of primary lung cancer. The complete guidelines accepted in 1976 are included at page 183.

Table IIB-20

Ontario Workers' Compensation Board
Lung Cancer Claims - Radon Gas Exposure

Year	New Claims Accepted	Cumulative Accepted	Cumulative Rejected
1970-1975	19	19	3
1976	7	26	7
1977	14	40	31
1978	9	49	47
1979	11	60	42
1980	20	80	40

A senior member of the Board's medical services staff has pointed out that in fact, one-third of the allowed cases demonstrated accumulated WLMs of less than 120, and that the Board routinely applied a 50% plus or minus factor in order to allow increased latitude in the evaluation of each case.[65] This may seem surprising to those who see the WCB as rigidly adhering to its guidelines. The profile of cases allowed up to the end of 1979 does indeed substantiate these figures - see Appendix 2. Three of these cases will be presented below to illustrate the application of this policy. (It is most unfortunate that this profile of cases was not maintained subsequently for comparison purposes.)

Since acceptance of the 1976 guidelines, a number of developments occurred which made it clear that a revision of the guidelines was essential.[66] First, health and safety issues had become an increasing focus of militant action and concern within the labour movement. Several meetings were held between Board officials and representatives of the United Steelworkers of America. In a formal meeting on June 26, 1979, the USWA and their consultants specifically requested that the reference to WLM be deleted from the guidelines for adjudication of lung cancer. Their argument was that methods of monitoring and obtaining WLM data, especially in the early years, must be considered as unreliable and that furthermore, as there is no threshold, any level of radiation may be a causative factor in the development of lung cancer.

Secondly, new scientific data had become available, principally through the work of Hewitt and Shannon for the Royal Commission on Health and Safety for Workers in Mines, by Dr. D.V. Archer and his colleagues at the National Institute of Occupational Safety and Health of the U.S. Department of Health and Welfare, and in testimony of various American and Canadian experts in the hearings held by the Environmental Assessment Board in 1978 and 1979 investigating the significance of uranium mining expansion in Elliot Lake. It became clear that a 120 WLM accumulation of radiation effect could not possibly be considered a threshold above which one could expect an increased incidence of lung cancer and below which one would not anticipate such an increase.

Guidelines for Adjudication
Lung Cancer - Uranium Miners

1. It is recommended that lung cancer for underground miners in the uranium industry be considered where any of the following circumstances apply:

   1.1 The employee worked at least 5 years underground prior to 1968 and the cancer is of the oat-cell type.

   1.2 The miner worked more than 10 years underground prior to 1968 and the cancer is primary in the lung.

   1.3 The accumulated radiation exposure is 120 working level months or more.

   1.4 Claims which do not meet the above guidelines shall be individually judged on their own merits and consideration given where it seems reasonably evident that the cancer resulted from uranium mining exposure. Consideration will be given to the variances in the record of radiation exposure prior to 1968.

   1.5 Borderline cases are to be considered on their own merits having regard for the accumulative radiation exposure and the benefit of reasonable doubt applies.

   1.6 The employee's underground work commenced after 1968 the case will be dealt with individually and consideration given where the radiation exposure records make it reasonably evident that the cancer resulted from uranium mining exposure.

APPROVED BY THE BOARD

January 5, 1976.

Uncertainty was said to have stemmed from lack of comparability of different mining groups (U.S., Canada, Sweden, Czechoslovakia) and most importantly, from lack of follow-up data in low exposure populations. The Royal Commission concluded that Ontario data supported a linear or non-threshold theory of radiogenic lung cancer and that significant risk existed well below 120 WLM of cumulative exposure, thereby inviting revision of the guidelines.

Besides pressure from labour, and the new data and recommendations of the Ham Commission, a third factor strongly argued for the revision of the compensation guidelines. The Atomic Energy Control Board had developed regulatory guidelines stipulating that accumulating 4 WLM in a given year or 2 WLM in any quarter of the year constituted the limit of an "acceptable risk". The AECB urged the WCB to remove the use of cumulative WLM from its adjudication guidelines in that it no longer indicated a threshold factor and might, in effect, have provided a false sense of security.[67]

The Board's Administrative Vice Chairman asked senior officials of the Medical Services Division to investigate the feasibility of removing references to WLM in the guidelines. A literature review was carried out and the conclusion reached that "reference to WLM levels could be removed from the guidelines, so long as consideration is given to other multiple factors which influence the onset of this disease in miners."

In August 1979, the Board approved new guidelines for the adjudication of lung cancer in radon and radon daughter exposure. (These are included in the text at page 186.) The new guidelines drop the 120 WLM criterion, delete reference to cellular type of tumour, eliminate the distinction of employment prior to versus post 1968, and adjust the latency period to a uniform interval of ten years from the previous five years for oat-cell and ten years for primary lung cancer.

With respect to the other parameters, there is still considerable debate as to the role of smoking. Radford suggested that in persons who develop lung cancer and had been exposed to radon gas, the causative factor should be considered the radon gas, despite the fact that they may be heavy cigarette smokers.[68] He also recently suggested that smoking has no effect on the latency period of radiogenic cancers, as mentioned earlier.[69] Archer, in contrast, suggested that smoking is a promoter rather than an initiator of lung cancer in individuals exposed to radon daughters, acting merely by shortening the latent period.[70] Another study contended that squamous cell cancers in uranium miners may all be cigarette-induced with radiation acting as a promoter.[71] Although the Board's pamphlet on occupational diseases erroneously implies that radiogenic cancer is unusual in non-smokers,[72] Board documentation cautions against overemphasizing smoking in evaluating a compensation claim.[73]

The significance of previous non-uranium mining exposure lies principally in that uranium is widely distributed in the earth's crust and may be encountered in many non-uranium mines; an estimate of this effect is in the area of one WLM per annum.[74] The thorium content in the ore may also contribute.[75]

In reference to the guidelines, one Board-employed physician urged that "latency of less than ten years should seriously be considered only if other variables such as exposure intensity, cumulative exposure and age at appearance of cancer are favourably placed. In no cases should latency less than five years be considered acceptable."[76] This remains the current basis for adjudication of lung cancer claims in uranium miners.

On April 3, 1980, a meeting was held between officials of the WCB and Ontario Hydro to discuss the establishment of guidelines for compensating malignancies in nuclear power plant workers. (It was stressed that the establishment of guidelines was the function of the WCB and that Ontario Hydro had no intention of attempting to dictate policies, but would provide expertise and required information.) Before basic criteria were brought forward, it became evident that type and site of cancer would have to be specified. The Board agreed to begin the process of developing guidelines which would be submitted for approval. Interestingly, the matter of genetic disturbances as it relates to birth defects was discussed, although it is not covered by the Act.

In September 1980, a report was submitted outlining the background, the approach in other jurisdictions, and the options. These are presented in full at page 187 as these options are potentially applicable to the other areas of industrial disease. The simple dilemma remains - we know that some cancers are caused by the workplace, but we still have no sound method of knowing with certainty which are the ones.

Guidelines for Adjudication Lung Cancer - Radon and Radon Daughters

1. Primary lung cancer caused by exposure to Radon Gas and Radon Daughters in industry shall be accepted as an industrial disease under Section 118, Section 1(1)(l) of the Act and Schedule 3 - Item 9.

2. Based on Ontario and other experience lung cancer claims in which the latency period is at least 10 years shall be considered using the following factors:

   2.1 Duration of exposure
   2.2 Exposure intensity
   2.3 Smoking history
   2.4 Geographical location of exposure
   2.5 Age when lung cancer first appears
   2.6 Year of entry into mining
   2.7 Age at start of exposure
   2.8 Previous underground exposure in non-uranium mining

3. Claims which fail to meet the criteria in Section 2, shall be considered on their own merits having regard to all factors. The benefit of reasonable doubt applies.

APPROVED BY THE BOARD
August 14, 1979

Options Re: Adjudication of Cancer in Radiation Exposure*

There are a number of alternate approaches available to the WCBO in the handling of claims for radiation related cancers. Some of these are listed below. All alternatives seem to have significant undesirable features.

Possible action:

1) Take no action. Continue to adjudicate claims individually as they arise.

2) Create rather loose guidelines which would accept:

    - myeloid leukemia cases on the basis of biological appropriateness of dosage and latency,

    - skin cancer in cases of radio-dermatitis or definite scarring,

    - bone cancers due to radio-ingestion on the basis of biologic appropriateness and dosage.

    - All other cancers would be rejected unless permitted dose under radiation protection regulations had been exceeded, except for lung cancer cases associated with alpha emitters which would be handled under the uranium guidelines.

3) Designate arbitrary exposures above which each specific cancer would be acceptable.

4) Designate some or all cancers as presumptively caused by radiation if exposure occurred, unless it could be shown to the contrary (as is the case with Schedule 3 diseases).

5) Adjudicate claims on a statistical risk basis, having regard to specialist epidemiologic advice from an expert in the field - in house if available, or from the Ministry of Labour or Radiation Protection Bureau in Ottawa.

It is suggested that further discussion with interested medical and claims personnel is desirable after a careful review of this report to determine if there is a consensus from which a policy recommendation could be developed.

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* Excerpt From: Chovil, Internal Report to the W.C. B., August 29, 1980.

3.1.3 Case Presentations and Discussion

Preamble: In the Industrial Disease Survey carried out for this study, there were 15 claims established for cancer due to radiation exposure. Six were actually claims set up due to over-exposure to radiation, but with no disease discernible. Of the nine claims for workers who had developed cancer, five were rejected, three were ultimately allowed, and one case was withdrawn pending the results of the Ontario Miners' Mortality Study. These cases are all presented below as illustrations of the application of the Board's policy.

To supplement this case list and provide a more balanced view, claims were reviewed from the case profile of allowed claims provided by a Board physician specializing in respiratory disease cases. Three of these were specifically chosen to present here as each was allowed despite failure to satisfy one or more of the criteria set out in the guidelines for adjudication. An additional allowed case was reviewed as, from the standpoint of labour, it provided a good example of its struggle to alter Board policy.

It should be kept in mind that as new scientific information becomes available and guidelines are revised accordingly, it is stated Board policy to review all previously rejected claims. In this sense, the present status of all claims should reflect current guidelines, regardless of the policy at the time the claim was initially adjudicated.

Case 1 - Claim 5: This worker died in 1974 of lung cancer at the age of 58, having worked in uranium mines 84 months between 1957 and 1964, accumulating apparently 68.3 WLM. He also had silicosis. A senior Board physician, on referring the claim to the Advisory Committee on Chest Diseases for their advice with respect to the silicosis, commented that "insofar as the lung cancer is concerned, I think the claim is rather shaky." In their response the Advisory Committee commented that while cancer claims are not normally referred to them, they wanted to record their observations with respect to the lung cancer as well as the silicosis. They noted that the figure of 68.3 WLM did not include any exposure during 1957 and they wondered as well, if the claimant's overtime was reflected in these figures. They felt that the lung cancer should, in fact, be compensable. A specialist employed by the Ministry of Health was consulted by the Board with respect to the low WLM submitted by the employer. He asked the employer to review and revise the estimates. After receiving no response, a senior Board physician who specializes in respiratory disease claims wrote "I do not think we can wait any longer. Based on the fact that he worked during the early dusty years, I think we should accept the claim." Consequently, the claim was allowed.

Commentary: This case begins to illustrate the inaccuracy and inadequacy of exposure information and the subjective assessment of the validity of the reported exposure. The Board doctor was hinting at rejecting the claim, whereas the Advisory Committee, only involved in this case coincidentally because the worker also had silicosis, drew attention to the questionable validity of the exposure record. Interestingly, it was the employer that was asked to verify the exposure rather than an uninvolved third party. In fairness, the fact that the claim was allowed does however indicate a certain flexibility on the part of the Board.

This case, as well as those which follow, raises the issue of whether the evaluation of exposure should rest in the domain of the medical staff, as opposed to that of industrial hygienists, or preferably, an interdisciplinary team.

Case 2 - Claim 6: This worker was 37 years old when he died of lung cancer in 1961. He had worked in the uranium mines for 45 months between 1958 and 1961, accumulating 114.7 WLM according to the Ministry of Natural Resources. (A 50% inaccuracy either way was noted). The claim was rejected in 1961 and reopened at the request of the family physician, the widow and the five children. In a letter to the family doctor in November 1974, the Board explained:

> The basic reason underlying the rejection was the short-lived period between his first exposure to radiation underground and the appearance of the oat-cell cancer. He had, in fact, only three years and nine months of such exposure. In his favour, of course, was the fact that he was very young and had an oat-cell cancer. In our experience, the average period in Ontario uranium mines has been over ten years from the time of first exposure to the appearance of the cancer, and this has included four or five cases of oat-cell cancer. The experience in the Colorado Plateau uranium mines suggests that a lag period as short as seven years could be accepted for oat-cell cancer.

The Acting Director of the Occupational Health Branch of the Ministry of Labour at the time, noted that the worker had accumulated 180 WLM between September 1956, when his employment began and 1960. Given this high level of exposure, his age and the fact that the tumour was oat-cell, he felt that the short lag was not sufficient reason for the rejection. On subsequent review, the claim was allowed.

The employer, however, contested allowance of the claim stating that the claimant did not have sufficient exposure to warrant allowance, insisting that he had only worked underground from 1956 to 1959 for a total of 35 months. The employer felt that the radiation levels it submitted should have been accepted rather than the much higher figures used by the Ministry.

In February 1976, the Board Consultant pointed out that this particular mine was a "hot mine" and given the age of the claimant, the oat-cell tumour, and a possible 50% error factor in the exposure calculation, the claim should be allowed. "Whether he spent three or four years underground is not the issue in this case: the issue is the sum total of events."

Commentary: The death of this 37 year old worker again illustrates the contentious nature of exposure information. As stated, the records of the company were clearly at odds with the records of the Ministry. The initial rejection of the case was on the basis that the lag time was much shorter than the average lag time shown in an epidemiological study. Recalling the limitations of epidemiology as discussed in part A of this chapter, this logic is most restrictive. The fact that the claim was finally allowed, indicates that the Board can be persuaded to be flexible.

Case 3 - Claim 7: This worker died in 1975 of lung cancer at the age of 52, having worked in uranium mines 60 months between 1958 and 1964, accumulating (according to the employer's records) 46.6 WLM. In January 1976, a Board physician specializing in respiratory disease claims wrote: "The WLM accumulation of 46.6 seems to be quite inadequate to accept a cause-effect relationship based on our approach to this problem to date wherein 120 WLM is used as a rough base-line." The denial, which was upheld by the Claims Review Branch, was appealed by the United Steelworkers of America in a letter dated August 20, 1976. Prior to the appeal hearing, the Board received a letter from the claimant's specialist stating that this man's lung cancer was very likely of the oat-cell type. The adjudicator, noting that records and measures to determine exposure were poor in those years, again referred the claim to the medical staff. By this time new guidelines had been approved and the Board consultant wrote "If we accept the oat-cell cancer, because this man has had five years' exposure his claim fully satisfies the criteria." The claim was thus allowed.

Commentary: This case, initially rejected, was allowed simply due to a revision of the guidelines, thereby attesting to the importance of the guidelines.

Case 4 - Claim 8: This worker was 46 years old at the time lung cancer was diagnosed. He began working in the uranium mines of Elliot Lake at age 26, working for a total of 71 months between 1958 and 1964 and accumulating apparently 64.8 WLM. In 1977, the diagnosis of lung cancer was made, providing a latency period of 19 years. It was noted that the lung cancer was of the squamous cell type. The Board doctor's comments were as follows: "It is felt that this claim does not meet the requirement as outlined in our guideline in that he has considerably less than 120 WLM and only has a total of 71 months exposure which is somewhat less than six years. Even giving this man the benefit of doubt the claim has no merit."

An appeal followed in which powerful arguments were put forward by the claimant's representative (M.P.P.'s Office) in criticism of the approach used to adjudicate this case, specifically with respect to the calculation of WLM. It was contended that the exposure estimate was grossly inaccurate and that indeed the claimant had accumulated over 120 WLM.

As is the usual procedure, after the hearing the documents were sent to the Board's medical consultant for comments. His comments, submitted in May 1979, proceeded to critique the submission, paragraph by paragraph. For example, while the representative pointed out that errors occur within a factor of 2, the Board's medical consultant stressed that this could mean that the claimant had only one-half the stated exposure. The representative pointed out the possible enhancing effect on accumulation of radiation exposure due to the presence of silica dust, and the consultant denied this insisting that only greater pulmonary impairment results, not synergism with carcinogenesis. This rebuttal misinterpreted the point that the claimant's representative was trying to make. Whereas the representative pointed out that the claimant worked an average of eight hours per week overtime and therefore, exposure should be calculated as 20% higher, the consultant insisted this was already reflected in WLM calculations which were based on an average figure for all individuals and that definite proof of overtime was not actually provided. The representative suggested furthermore, that the claimant's job as a diamond driller put him at greater than average risk, but the consultant pointed out that this was speculative as no scientific evidence of this effect was available. The claimant referred to situations in other countries, while the consultant pointed out that mining conditions were not the same as in Ontario. Whereas the representative suggested that the Board was negligent in not verifying the accuracy of WLM estimates provided, the consultant retorted that the Board never had any reason to doubt the validity of the figures within the limits of accuracy dictated by the existing technology.

The Board's medical consultant did agree that the representative had a valid argument with respect to the possible risk at low level:

> It has been alleged that even one WLM includes a little extra risk, but clearly the probability of a cancer developing at this level becomes so small as to be without any significance. As the cumulative exposure increases, the probabilities in any one case of cause and effect rise. Since not all lung cancers can be considered occupational, we are faced with a judgment decision in identifying a level of cumulative exposure where the probabilities can be dealt with by the Board in a credible fashion. Based on the evidence, we choose 120 WLM as a level above which all cases would be accepted (with reasonable latency), but not (as a floor) below which no cases would be acceptable. With prolonged latency we have accepted claims in the 70 and 80 WLM level and even less than that where the cancer is oat-cell.....the Medical Branch assessed this claim within the structure of the guidelines accepted by the Board. While the guidelines are subject to change, and this claim would conceivably become acceptable in the future, we felt that we were not able to anticipate such changes by accepting the claim as it now stands.

He also suggested that better effort be made to define exposure.

In September 1979 the claim was reviewed following the approval of new guidelines. The same Board consultant now expressed the case as follows:

> This man was 27 years old when he started mining in Elliot Lake. He spent six years underground. He had been a heavy smoker all his life. He was 46 years old when squamous cell cancer was discovered and had accumulated 65 WLM at the time. He worked four years in other Ontario mines for an additional 4 WLM.....The exposure here places the case in the mid-30 group examined by the Royal Commission - average WLM 58, average exposure 4 1/2 years and average latency of 12 1/2 years. There were radiogenically derived cases in this group, and the claimant exceeds the average.....As a heavy smoker, smoking induced lung cancer would not be expected to appear at such an early age, the influence of radiation exposure therefore must be considered. The lag period of 18 or 19 years is consistent with the age of start of exposure. The latent period seems to fit the postulated synergism between smoking and radiation. All in all the amount of exposure, the latent period and the age at which cancer was diagnosed suggested that radiation may have played a significant part in the genesis of lung cancer. Therefore, under the new guidelines the claim should be acceptable.

The claim was consequently allowed.

Commentary: Again illustrating the importance of the guidelines, this case depicts a number of other important issues. Aside from the content of the arguments put forward by the Board doctor concerning the points raised by the claimant's representative, it should be noted that these remarks were made in the form of an internal critique of the evidence presented at the hearing, with no opportunity for clarification or rebuttal by the worker's representative. The attitude taken by the Board doctor was adversarial in nature, siding against the claimant where there was possible doubt. Several of the Board doctor's statements such as "the Board never had any reason to doubt the validity of the figures..." seem to contradict previous decisions regarding exposure assessments.

Case 5 - Claim 9: This worker died of lung cancer at the age of 59. He had spent 36 months in an Ontario uranium mine between 1957 and 1960 and another ten-odd years underground in the non-uranium mines in Ontario. His working level months were estimated at approximately 50. The claim was rejected in 1977 and appealed. In April 1978, the Board consultant wrote: "We feel that we should confirm the rejection of the claim as it does not meet the criteria laid down in our current guidelines. It is further noted that the diagnosis was carcinomatoses and that the lung was suspected as the area of the primary but this was never proven." The claim was reviewed in 1979 due to the new guidelines. The Board doctor noted: "He was 59 when he died and this would therefore subtract from the merit of the claim since in a smoker lung cancer could be expected to appear in this age group frequently." However, this time he noted: "This is a rather borderline claim.....There is a probability of some contribution by radiation to the lung cancer, therefore it should be considered compensable." The claim was thus allowed in January 1980.

Commentary: Factors that were referred to as detracting from the merit of this case were the lack of precision in diagnosis, borderline exposure and the fact that the worker was a smoker. (It could be noted that the exposure in non-uranium mines was not taken into account.) Nonetheless the allowance of this case after revision of the guidelines does indicate some flexibility on the part of the Board, and again illustrates the importance of the guidelines.

Case 6 - Claim 10: This worker died of lung cancer at the age of 54. He had worked in numerous mines in Quebec, Manitoba and Saskatchewan as well as Ontario. There was some discrepancy as to how many years he actually spent in the uranium mines of Elliot Lake. The employer stated that he worked underground in Elliot Lake from 1958 to 1961 only, the case summary referred to a "five-year stint" in Elliot Lake and the final adjudication listed his exposure in Ontario uranium mines as 25 months between 1958 and 1968. His Ontario WLMs were recorded in the Statistical Analysis Division of the Board as 12 in 1959, 5.4 in 1960 and 1.48 in 1968 for an estimated total of 18 to 20 WLM. The latency period from first exposure in uranium mines was nonetheless 20 years. It was also noted that he had been a very heavy smoker. The claim was rejected and again upon review, subsequent to the new guidelines in 1979, it was noted that "this cancer could be easily explained by a smoking history alone and the exposure in the uranium mines here was brief." The rejection was thus reconfirmed.

Commentary: A key issue in this case was that the worker's exposure outside Ontario was not taken into account; his being a smoker was also a major factor in the rejection. With respect to the first point, it should be noted that the policy, for example, in silicosis, is that the last exposure employer bears the loss. In this regard and especially in considering the extreme mobility of miners, it is contentious that exposure outside Ontario is discounted. With respect to the second point, the fact that the smoking history could have accounted for the cancer is not necessarily relevant.

Case 7 - Claim 11: This worker was 55 years old at the time his lung cancer was diagnosed. He had been employed at a uranium refinery for 25 years and it was noted that he had been exposed to radiation at least since the age of 25. The Board ruled that he had received a total of only 25 WLM exposure and the claim was therefore denied. It was originally appealed in July of 1979 at the request of the United Steelworkers of America, a second appeal was requested. The union submitted various articles from the scientific literature and systematically proceeded through the criteria of the guidelines, posing questions on the manner in which information in each was applied to the claimant's case. With respect to the duration of exposure for example, it was rhetorically asked during what periods in the 25 years of exposure was the claimant not exposed to radiation; with respect to exposure intensity the enquiries focused on the accuracy of radon concentration measurements and whether or not gamma exposure was included in the estimate as well as the specificity of these estimates to the claimant's actual working area. The representative wondered if the Board had taken into account the nature of the claimant's job which consisted of an open process of dumping feed material containing a fair amount of radiation into a drum. He wondered if radon, radium and other daughter products were calculated and whether exposure during the cleaning process had been taken into consideration. He also pointed out that it is generally accepted that no threshold exists for radiation-induced cancer. As to the claimant's smoking history, he questioned the nature of the scientific literature. As well he questioned the information available to the Board concerning the amount the claimant actually had smoked. With respect to the age when lung cancer first appeared, he pointed out the claimant was 45 when the cancer was discovered and that exposure beginning at age 25 provided a latency period well in excess of the ten years required in the Board guidelines. He referred to the worker of Claim 12 (see Case 8 below) who worked in the same area as this claimant. He pointed out that the urine analyses apparently indicated that this claimant's uranium dust ingestion exceeded that of the other worker, however, the Board allowed the claim for the latter and rejected this claim. Lastly, the union representative pleaded that if there was a threshold and if the records were accurate to show that the claimant's exposure was below the threshold, then he would agree to the rejection, but with no accurate records regarding exposure and no threshold, the benefit of doubt should apply.

The employer's solicitor argued that nothing in what was presented constituted new evidence and therefore, the previous rejection should hold. A 139-page brief was submitted by the employer containing a review of the claimant's lost time injuries, a summary of minutes from union-management meetings, a respiratory report, government occupational health reports and a radiation exposure history for the claimant.

The Board decision, rendered September 1981, stated "...no new evidence or argument was presented at this hearing which would cause the Appeal Board to vary, amend or revoke the Appeal Board decision of July, 1979. Further, the Appeal Board finds that this is not a case where the benefit of reasonable doubt could be applied."

Commentary: The arguments presented by the claimant's representative revealed many of the weaknesses in the adjudication of cases of this nature, specifically with respect to accuracy of exposure analysis and records, and again highlighted the threshold issue. It should be noted that the employer played a prominent role in contesting this case, presenting an extensive brief to support rejecting the claim. Despite the inaccuracies in

WLM measurements, the Appeal Board decided that the benefit of the doubt could not be applied.

Case 8 - Claim 12: This man worked as a maintenance mechanic at an Ontario uranium refinery from 1948 to 1966. Although there was considerable variation between the claimant's description of his job and that given by the employer, the latter listed his cumulative radiation exposure as 82 WLM noting that the error in its estimates may be in the order of 50%. His lung cancer was diagnosed as being of the oat-cell type. The Board consultant wrote:

> It is felt this claim meets the requirements as it exceeds the minimal five-years mentioned in our guideline for the allowance of an oat-cell cancer. As well, this man has had an exposure interval from 1948 to 1966 representing 18 years and an inception period from 1948 to 1977 of 29 years. Therefore allowance is recommended.

The claim was allowed.

Commentary: This case serves essentially as a contrast to the previous claim from a fellow worker. It raises the issue of "where to draw the line." Whereas 85 WLM was considered acceptable, 25 WLM was not.

Case 9 - Claim 13: This worker was 52 years old in 1979 when his lung cancer was diagnosed. He had been exposed to radiation from 1959 to 1970 in Elliot Lake mines for 134 months of exposure in which he accumulated a WLM of 62.8. The Board doctors noted that he had a latency period of 20 years, a cessation interval of nine years, was 52 when his cancer was diagnosed and was 35 years old when first exposed to radon daughters. It was felt that this claim met the requirements and therefore it was allowed in January 1980.

Commentary: This case illustrates that a cancer can be felt to be work-related without having to meet a specific exposure requirement, and serves as contrast to the claims rejected due to insufficient exposure, discussed earlier. Note that this case was adjudicated after all quantitative stipulations, other than the 10-year latency requirement, had been dropped.

Case 10 - Claim 14: This worker was 52 years old at the time his nasal cancer was diagnosed. He had been employed in the Elliot Lake uranium mines from 1958 to 1964. The claimant's specialist wrote the Board stating that "since the claimant had been exposed to radioactive dust, it is difficult to justify non-acceptance of this claim." A Board physician who reviewed the case however felt that "there is no evidence to show that exposure to radioactive dust would cause nasal cancer." He advised the Appeal Board that a study was presently being undertaken by the WCB in conjunction with the Ministry of Labour as to the relationship between cancer and uranium mining. The claimant was informed that it would take at least two years before results were obtained. The Appeal Board gave the claimant the choice of (a) await results of the study, (b) have the Appeal Board render a decision on the evidence on file or (c) withdraw the appeal and have a new hearing when the results of the study were known. The worker advisor handling the claim discussed the options with the claimant and the claim was withdrawn.

Commentary: Clearly, the length of time required to complete an epidemiological study is incompatible with this worker's need for compensation. Perhaps a different approach by the claimant's representative might have altered the outcome in this case. It may be noted that the opinion of the claimant's specialist regarding work-relatedness was ignored. It is also of interest that in other jurisdictions, Newfoundland, for example, nasal cancer in a worker exposed to radiation is indeed considered compensable (based on the studies done in that province noted earlier). If all workers in Ontario have to await Ontario-based epidemiological studies before being granted compensation, the number of compensable work-related cancers will remain miniscule.

Case 11 - Claim 15: This worker filed a claim for cancer of the larynx. He had worked underground in Elliot Lake from 1958 to 1968 for a total of 120 months of exposure. His WLM was estimated as 70.8. Although he had an adequate history of exposure and latency period, his cancer was of the wrong site and therefore, the claim was rejected.

Commentary: Due to the limitations in epidemiological methods discussed in part A of this chapter, it is often difficult to show an increase in all types or sites of radiogenic cancer unless the excess is exceedingly large. Notwithstanding the fact that different tissues have different degrees of sensitivity, the rejection of a claim due to lack of evidence of an excess cancer rate at this particular site in an individual with adequate history of exposure to his respiratory tract and appropriate latency period may well be unduly restrictive. (In this case as well the claim would have been deemed compensable in Newfoundland.)

Case 12 - Claim 16: This worker developed lung cancer having worked as a radiographer from 1939 to 1955. As the dosimetry service only originated in 1952, only 322 mrem of radiation exposure was recorded. It was felt that although employment involved gamma radiation exposure, there was little possibility of significant alpha exposure. A Board-employed physician noted that although skin cancer and leukemia were well established in this occupation, there was no real evidence of a greater incidence of lung cancer in this group, compared to the general public. The claim was rejected.

Commentary: In contrast to the previous case where the issue was wrong site of cancer, here the issue is wrong type of radiation. (The very small dose of radiation exposure actually recorded may also have contributed to the rejection.) Referring to the scientific background in Section 3.1.1. it could be argued that there is enough controversy surrounding this issue to raise the 'benefit of the doubt' argument.

Case 13 - Claim 17: This worker was 40 years old when Hodgkins disease was diagnosed. He had over 20 years' exposure in uranium mines, including an employment of 6 1/2 months in 1957 and continuous employment from 1959. It was specifically noted that he worked as a driller for 15 years in the raises. The claimant contended that there was a relationship between his Hodgkins lymphoma and gamma exposure. Although the employer denied any significant gamma exposure, the union contended that gamma rays were present especially in close confinement areas such as those locations where the claimant had worked. He produced a letter from the Atomic Energy Control Board stating that results of studies by Board staff and by the uranium mining companies of Canada, the U.S. and France indicated that employees of uranium mines are likely to be exposed to significant doses of gamma radiation. (No specific reference to Elliot Lake Mines was made). The letter also indicated that the target date for implementation of a personnel dosimetry program in the Elliot Lake Mines to measure gamma exposure was December 30, 1980. The union representative acknowledged that the relationship between the Hodgkins and gamma ray exposure has not been proven, but felt that the benefit of doubt should be resolved in favour of the claimant. The Board doctor pointed out that gamma radiation in the Colorado studies could have been substantial, because of the lack of ventilation and the richness of the ore as compared to Elliot Lake. He noted that the Ham Commission concluded that gamma exposure in Elliot Lake was within the permissible level and that in all the literature devoted to the analysis of mortality associated with uranium mining in Canada, the U.S. and Europe, there were no reports of increases in tumours of lung tissue, either blood borne as in leukemia, or solid as in Hodgkins. The claim was denied.

Commentary: Some of the same issues raised in the previous cases are once again illustrated here. Aside from the question of the debate in the scientific literature concerning types of cancer that can be caused by radiation exposure, the extent of exposure was again contentious. It is noteworthy that the Board Consultant referred to the Ham Commission's observation that gamma exposure was within the "permissible level". From the preceding discussion it is clear that "permissible level" is not synonomous with "safe level" and that the evidence suggests that there is no "safe level" for radiation exposure (see Section 3.1.1.) Although it has some bearing on the probability of developing the disease, and is therefore important in preventative efforts, reference to these regulatory levels is of less relevance to the adjudication of a case of an individual who has indeed contracted the disease in question.

General discussion

It can be seen clearly that the guidelines do indeed play a decisive role in the adjudication of claims. Although flexibility is applied in weighting the different criteria in the guidelines, as shown in Cases 1 and 2 which were allowed despite failure to meet one or more of the criteria, the mere changes of guidelines were shown to significantly change the approach to the facts in the claim. This was shown in Cases 3, 4 and 5 which were initially rejected, and subsequently accepted when guidelines were altered. Cases 6 and 7 were rejected due to insufficient exposure, despite adequate latency periods and correct tumour site. Case 9 met the guidelines for a uranium miner with lung cancer and therefore, was swiftly allowed.

Adequate scientific evidence linking the tumour site to the type of radiation in an epidemiological study seems to be of paramount importance, as was shown in Cases 10 to 13. In case 10, a uranium miner who developed nasal cancer, withdrew his claim pending receipt of the Ontario Miners' Mortality Study results. Case 11, the claim for a uranium miner who developed cancer of the larynx, was denied despite the long history of radiation exposure to his respiratory tract and adequate latency period. Case 12, the claim for lung cancer in a radiographer and Case 13, the claim for Hodgkins in a uranium miner were also denied due to the Board's finding that the claimant had the 'wrong' type of radiation exposure or developed cancer at the 'wrong' site to make a cancer claim allowable.

These thirteen cases summarize the difficult issues in the adjudication of cancer claims in workers exposed to radiation. These issues are not peculiar to radiation exposure, and as will be shown below, have their parallels in the adjudication of other cancer claims.

3.2 Cancers Caused by Soots, Tars and Oils (Polycyclic Aromatic Hydrocarbons)

Cancer claims involving exposure to polycyclic aromatic hydrocarbons (PAHs) are the subject of three separate sets of guidelines at the Ontario Workers' Compensation Board and have accounted for 61 allowed cancer claims as of December, 1981: 12 for cases of lung cancer in coal gasification workers, 16 cases of lung cancer in workers exposed to coke oven emissions, and 33 cases of lung cancer in workers exposed to foundry environments. (It is not confirmed that PAHs are the sole causative agent here). Numerous cases of skin cancer due to exposure to coal tar pitch have also been accepted.

There is a wealth of information concerning the effects of this group of chemicals on various tissues and organs of laboratory animals. In humans however, exposure is not to individual chemicals, but to combinations as they occur in soot, coal tar, pitch and mineral oils as well as tobacco smoke and exhaust. Occupations in which exposure to such substances occur are numerous.

In Ontario, exposure to carcinogens in coke oven emissions has been the focus of considerable concern and the Ontario Government has recently established regulations for coke oven emissions as a designated substance under the Occupational Health and Safety Act. Concern about cancer in foundry environments has also been expressed, as has concern with respect to exposure of various workers to mineral oils, such as those used for lubricating and as cutting oils. Particular difficulty in this area arises largely because no single carcinogenic substance has been identified as the causative agent responsible for the increased risk in the various exposures. Although much data exists on individual PAHs, the exact mix and physical properties of PAHs varies considerably in different compounds, in different processes and even within the same process under different situations. Not all substances in this group of chemicals are equally carcinogenic, and some, in fact, do not seem to be carcinogenic at all.

As will be shown below, due to the heterogeneity of these chemicals and the industrial processes in which they are found, data on the chemicals as a class or even on individual substances are largely ignored by the Compensation Board in favour of epidemiological evidence of increased cancer risk in specific occupational sub-groups. As is the case with occupational carcinogens in general, this approach leaves much to be desired. Controversy in this area as in others focuses largely on the extrapolation of the results of various epidemiological studies to a specific case in question.

Some historical and scientific background will be presented so that the basis of the controversy can be appreciated. This will be followed by a description of the evolution of policy at the Ontario Workers' Compensation Board with respect to the adjudication of cancer claims resulting from exposure to these chemicals. Cases will then be presented to illustrate the decision-making process and the current debate.

3.2.1. *Scientific background*

In 1775, Percival Pott attributed the occurrence of cancer of the scrotum in chimney sweeps to their occupational exposure to soot. As new industries became established, other industrial carcinogens (coal tar, paraffin wax and certain mineral oils) were recognized, at the expense of human tragedy. Finally, it was found that coal tar, a proven carcinogen in man, could evoke a carcinogenic response in rabbits[77] and in mice.[78] It has now also been shown that tar, pitch, and mineral oils are carcinogenic not only to the skin, but to internal organs as well.[79] The finding that it was possible to induce similar cancers in animals resulted in worldwide research to isolate the active carcinogenic constituent.

Studies soon showed that the carcinogen in these compounds was probably some complex aromatic hydrocarbon[80] (a chemical with a structure consisting of one or more carbon rings akin to benzene - see diagram in Appendix 3a). Benz(a)anthracene was the first recognized carcinogen of defined chemical constitution, and benzo(a)pyrene followed shortly afterwards. It soon became clear that carcinogenic activity was not confined to just a few types of chemical structures. As screening in experimental animals became increasingly expensive and lengthy, attention increasingly turned to trying to understand the mechanism of carcinogenesis with the hope that carcinogenic potency could be recognized on the basis of chemical structure.

It is now known that carcinogenesis induced by aromatic hydrocarbons involves at least two stages, initiation and promotion as discussed in Part A. Initiation is the only stage which absolutely requires the presence of a carcinogen. It has been shown that if a sufficiently large dose of carcinogen is administered, tumours will arise even in the absense of any promotion. Even for those PAHs shown to be carcinogenic, it cannot be assumed that they are all equally potent initiators or equally effective promoters[81], and some PAHs are thought, in fact, to be anticarcinogenic[82]. Since the 1930's, a large number of PAHs have been screened for carcinogenic activity and much information now exists on the relative carcinogenicity of chemicals with different structure.[83] (A summary of the findings for a few PAHs appears in Appendix 3a). General concern has been expressed regarding the limited experimental evidence upon which the "non-carcinogenic" status of many PAHs depends. Recent re-examination of "inactive" compounds revealed that several such compounds exhibit initiating activity, even if they have not been found to be complete carcinogens.[84]

Benzo(a)pyrene is known to be one of the major constituents of soot. Domestic soots are the products of imperfect combustion of carbonaceous materials such as wood, coal and oil, and industrial soots (carbon black for industry) are produced by the incomplete combustion of natural gas or oil residues. Today exposure to domestic chimney soot is not significant as chimney sweeps no longer climb inside chimneys. However, exposure to carbon black occurs among its manufacturers and users, notably rubber and paint makers. No increase in cancer risk has yet been demonstrated among workers, although cases have been described in the mixing of carbon black and oil in the rubber industry.[85]

Coal tar and pitch are the residual products of coal distillation and have long been associated with an increased risk of skin cancer.[86] Exposure occurs mainly in the coal, gas and coke industries, in steelmaking plants, and in the manufacture of patent fuels. Cases of skin cancer attributable to exposure to pitch have also occurred among optical lens grinders, electrical equipment workers, wharfmen, cable layers, net fixers and fabric proofers.[87] Exposure to creosote oil occurs among brick and tile workers, as well as among timber proofers.[88]

In 1947 Kennaway described an increased frequency of lung cancer among workers exposed to coal, gas and tar.[89] Doll examined the mortality experience of British gas workers, finding a significant excess of deaths from lung cancer, as well as an elevated rate for bladder cancer and cancer of the skin and scrotum. No substantial evidence of hazard among the by-product workers was noted at the time.[90]

As early as 1936, it was noted in Japan that coke workers and workers exposed to coal tar fumes in generator gas plants were at increased risk of lung cancer, with the increase paralleling the length of exposure. In this study the increase was demonstrable beginning ten years after initial exposure.[91] Since then, much more information has accumulated regarding the cancer risk to steelworkers.

Cancer and Coke Oven Emissions: Steel companies operate coke ovens to convert coal into coke which in turn, is used as a fuel for making the iron in blast furnaces. It is now widely agreed that there are volatile carcinogens in the vicinity of coke ovens. These substances are produced when the coal is baked at high temperatures in the absence of air, necessary precisely to drive off most of the volatile material which constitutes approximately 30% of the weight of the coal.

In 1962, the University of Pittsburg School of Public Health, with the co-operation of three major steel companies, launched a comprehensive survey of steelworker mortality. Under the direction of Dr. J. William Lloyd, the health records of some 59,000 workers employed in seven steel mills in Allegheny County were tracked down. The study was later expanded to include workers at ten other steel mills. The findings of this national survey coincided with those of the original: coke oven workers as a group are 2 1/2 times as likely to die from lung cancer as steelworkers as a whole. The lung cancer risk depends in part on length of exposure and exact location of the work. For instance, after five years on the job, the lung cancer death rate rises to 3 1/2 times the expected but for top-side coke oven workers alone, the rate is 7 times the expected. For top-side workers with at least five years on the job, the rate rises to 10 times the expected. An unexpectedly high death rate from kidney cancer was also noted (7 1/2 times the expected). Variables such as smoking and geographic location were found not to be able to account for the results. (These studies were presented in a series of 10 articles in the *Journal of Occupational Medicine.* Tabulation of results from some of the studies[92 - 97] appears in Appendix 3b.) Emissions from coke ovens in the U.S. were analyzed for PAHs in 20 coke plants and although their levels varied in each plant, similar ratios between benzo(a)pyrene, chrysene and benz(a)anthracene were found.

As far back as 1953, Dr. Carol Redmond of the University of Pittsburgh had been gathering data on several sub-divisions of the coke plant, particularly, workers employed in the non-oven areas. It is noted that classifying workers according to whether they worked in the coal handling area, the coke ovens or the by-product plant is often difficult, as job titles do not indicate the amount of time spent in each precise area. For purposes of the Allegheny County study, all workers who held jobs in both oven and non-oven areas were classified as coke oven workers, although this tends towards under-estimating the actual coke oven risk. The coke oven workers were further sub-divided into full-time topside and side oven workers, and the non-oven workers sub-divided into by-product plant and coal-handling (see Appendix 3b for table of results). All coke workers with five or more years of experience in the coke plant had excesses of mortality due to lung cancer. As well, rates of cancer of the kidney and prostate were elevated in both oven and non-oven workers. Cancers of the digestive system were noted to be significantly elevated in non-oven workers, particularly cancer of the colon and pancreas. It was also noted that cancers of the buccal cavity and pharynx appeared to be elevated. Redmond stressed the need to consider non-oven as well as oven workers when evaluating cancer hazards in the coal plant[98].

Dr. Edward Radford of John Hopkins University studied coke oven workers in plants not included in the Lloyd and Redmond[99] studies. Results suggested that cancer of the respiratory system, bladder and kidney were elevated and that these excesses were not restricted solely to coke oven workers, but generally to all workers in the steel industry.[100] (The series of steelworker mortally studies was published as a set in July 1981 by NIOSH, reviewing and confirming these findings.)[101]

Thus various studies have linked cancers of various sites, including respiratory, digestive as well as urinary system cancers, to exposures in the steel industry. The studies varied from noting the increase in top-side coke oven workers alone to coke oven workers generally, to all non-oven workers in coal plants to even all workers generally in steel mills.

An assessment of the significance of the results must be made on appraising the strengths and weaknesses of various aspects of epidemiological methodology and statistical testing. Suffice it to say that while the carcinogenicity of exposure to PAHs in the steel industry is unquestionable, the relative magnitude of the risk at various worksites is still largely uncertain.

Cancer in Foundries: In recent years it has become apparent that lung cancer is indeed a hazard associated with foundry exposure. Although the exact relationship between the risk of cancer and exposure to specific pollutants in the foundry has not been established, PAHs such as benzo(a)pyrene have been found in many foundry locations, as well as chrysene and benz(a)anthracene. These are produced as pyrolysis products of organic materials used in mould binders and additives and are prevalent during mould and core-making operations, as well as pouring, cooling and shake-out. (Besides PAHs other classes of carcinogens present in foundry environments include nitrosamines, aromatic amines, carcinogenic metal salts and physical carcinogens. It has been suggested, for example, that the interaction between airborne PAHs and metallic particulates may be important.[102])

The first reports of excess lung cancer among foundry workers were issued in England in 1938 based on census population figures and official death records.[103] Another indication of elevated lung cancer risk was noted in autopsy studies.[104] In 1979 Egan et al. using the death records maintained by the International Molders and Allied Workers' Union, reported excess risk of cancer of the lungs, and the suggestion of excess cancers of the buccal cavity and bladder.[105]

The first lung cancer mortality studies in foundries to employ the retrospective cohort mortality method was performed in Finland. Here it was found that individuals who were employed for less than five years in typical foundry occupations experienced a lung cancer mortality rate only slightly greater than expected (1.3 times the expected rate), however, the lung cancer mortality rate among workers exposed for more than five years was 2.1 times the expected value. In this study, elevated lung cancer rates were observed only in iron foundries with no excess risk noted in steel foundries.[106] Interestingly, in Lloyd's studies no particular excess mortality for foundry workers was noted, although it seems that a possible link with lung cancer did indeed occur. Lloyd did however comment on the large number of respiratory cancer deaths occurring in a community living downwind of a steel foundry.[107] A study of iron foundry workers in Finland in 1978 also revealed a statistically significant excess of lung cancer, this time in workers with at least one year of exposure.[108]

Of major significance to the worldwide literature on the subject and particularly to the Workers' Compensation Board of Ontario was the work of Gibson et al.,[109] who conducted a ten-year retrospective study of lung cancer deaths in the workforce of a Hamilton foundry. This study indicated that foundry workers with five or more years of exposure had a greater lung cancer mortality rate than a group of non-foundry workers from an adjacent steel mill. Risks varied according to the job performed, with crane operators having the highest risk, followed by fettlers, moulders and core makers. There was no significant difference in the mortality rates for cancer at sites other than the respiratory tract. (See Appendix 3c for tables of results). The details of this study and the sequence of events that followed will be discussed in detail below.

In a recent study of mortality patterns among workers in a U.S. iron foundry, a twofold excess in mortality from both cancer of the digestive system and respiratory tract was reported.[110] The investigators noted that the small number of workers (192 men) in this group limited the chance of detecting a real increase in cancer risk and that additional studies were necessary. As was pointed out in a recent literature review on the subject,[111] in most of the epidemiological investigations cited above the study populations were relatively small and in many cases the number of cancer cases available for study were too few to test for statistical significance.

Current work in several countries is addressing the problem of quantifying actual foundry exposures. An important study in this area, commissioned by the Workers' Compensation Board of Ontario, has just been published and will be discussed below.[112] Interestingly, although correlations between levels of PAHs and cancer risk were demonstrated in the Finnish studies, there was little correlation between epidemiological data and analysis of air samples in the Hamilton foundry study. For example, the total quantities of PAHs were highest in the furnace area where the lung cancer risk was minimal. However, it was noted that epidemiological data reflect exposure of thirty years ago and therefore it may not be valid to attempt correlations with current exposure levels.

Thus, here again, the exact magnitude of the cancer risk, the specific sites targeted and the various risks associated with the various types of foundries are not entirely clear. Nonetheless, it is indeed evident that there is an increased cancer risk associated with foundry work.

Mineral Oils: In 1875 scrotal cancer was described in workers producing paraffin by coal tar distillation[113] (later found to be due to the oil produced)[114] and the following year increased scrotal cancer was found in Scottish shale oil workers. Scrotal cancer in the cotton mule spinning industry was noted to be a severe problem[115] and resulted in the enactment of regulations in 1953 in Britain restricting oil usage in mule spinning.

Cutting oils used by workers to cut metals were found to increase the risk of skin cancer in England[116] and toolsetters and workers in setter operations in automobile shops who use heat-cutting oils have been shown to have an increased risk of cancer. Metal rolling, tube drawing, metal hardening and machine operating are also associated with increased risk. Workers have also been affected from exposure during changing of transformer oil in electrical substations and during the painting or spraying of mould oil for brick and tar-making or concrete moulding, drop forging, rubber mixing, wire drawing, rope making and from grease and metal working.[117]

Cancers of the larynx, lung and stomach have also been attributed to oil mist[118] and recent evidence has shown that persons who develop cancer of the scrotum are significantly more liable to develop cancer of other sites, for example, the respiratory tract or upper digestive tract.[119]

Mineral oils vary in their carcinogenicity and their composition, however carcinogenic oils include even those which lack benzo(a)pyrene. Generally the carcinogenicity of these oils increases as the temperature is increased. Studies have produced conflicting results and the extent of carcinogenicity in this group of exposures is still not well known (other than those containing nitrosamines).

In summary, the cancer hazard of polynuclear aromatics which occurs in coal-coke exposure has been known for centuries and much animal and chemical data exists about this class of carcinogens. As exposure patterns vary substantially in various occupational groups, the extrapolation of epidemiological findings from one group of workers to another is very tenuous and there is even reluctance to generalize from one plant to another.

Some studies have shown that steelworkers as a whole have increased cancer risk (not merely coke oven workers). Other studies were only able to demonstrate this increased risk in workers in the coke oven plants. It is known that topside workers are at greatest risk, that oven workers are also at increased risk compared to the general population, as , in fact, are non-oven workers in the coke plant.

Cancer in sites other than simply the lungs has been shown to be associated with exposure, including cancer of the kidney, bladder and prostate, colon and pancreas, and likely cancer of the buccal cavity and pharynx (including laryngeal cancer).

No precise information regarding the interaction of smoking and coke oven exposure has yet been produced, as smoking histories are difficult to obtain in studies of this nature.

With respect to foundry exposure, some studies documented an increased risk in iron foundries only, while further studies have confirmed that steel foundries do, indeed, also have increased lung cancer risk. Some studies have shown that this risk is not confined to lung cancer alone, but is present for other sites as well, particularly the digestive tract. The study populations have been too small to state with a high level of certainty at what length of exposure and period of latency this risk becomes manifest. Gibson's study of Hamilton foundry workers has been of particular importance in Ontario, as will be discussed below. As the causative agents are still cause for considerable speculation, the possibility of associating increased risks with estimated levels of exposure is completely out of the question.

Workers exposed to cutting oils may also have an increased risk of cancer at various sites, however, the large number of possible mixtures of chemicals involved and the variety of exposure patterns make any sweeping conclusions regarding these oils very tenuous. No good epidemiological evidence exists at all in this area.

Despite the uncertainties, the mountain of evidence linking occupational exposure to PAHs with increased cancer risk is undeniable. The development of policy by the WCBO to deal with claims from workers who developed cancer in these settings was therefore necessitated.

3.2.2 *Evolution of Board Policy*

The first recognition by the WCB that these chemicals were carcinogenic occurred early in its history, and cancer due to exposure to coal tar pitch was placed in Schedule 3 of Diseases in 1932. In 1942 it was clarified that the presumption of Schedule 3 only applied to skin cancer and to certain processes in which exposure to tar, pitch or oils occurred. Studies of coal gasification workers were conducted in Ontario resulting in the allowance of twelve lung cancer claims from workers engaged in coal gasification in the late 1940's, thereby establishing an historical precedent.

Based on the suspicion of an increased lung cancer risk in U.S. steel plants, a study was begun in Ontario in the early 1960's by the Ministry of Health. Due to the lack of records the study had to be aborted.[120] However, based on statements from the plant's Safety Compensation Officer at a large steel company, it was felt by the WCB that coke ovens in Ontario do not really differ from those in the U.S. and the American experience should be accepted here.[121] By 1974 claims had begun to be submitted and by April 1975 the first recommendations for adjudication criteria were put forward.

Based on the Board's analysis of the literature, various conclusions were drawn. One assumption was that coke oven workers at ground level were not at significantly increased risk, and therefore, a lung cancer claim from a 50 to 60 year old, particularly in a smoker, would not have much merit. It was also concluded that topside workers required five years or more exposure and a ten-year latency period, and bench level workers aged 40 to 50 with ten to twenty years exposure in old ovens would merit consideration, as would oven workers aged 45 to 55 with ten to twenty years working partly on the ground and partly at bench level. Lung cancer in the 60 to 80 age group would be compensable only with twenty to thirty years at ground and bench level and in an oven of old design for a non-smoker; for a smoker, exposure of twenty to thirty years at bench level and topside would be required. It was mentioned that these criteria would be subject to comment by the Department of Health and the companies involved.[122] No input from labour organizations was requested.

The inclusion of smoking as a factor in the assessment of lung cancer risk in coke oven workers was defended by a senior Board physician in December, 1975 in that benzo(a)pyrene and other carcinogens found in coke oven emissions were also found in tobacco, that animal evidence showed that the same skin cancers produced by coal tar are also produced by tobacco, and that tobacco also contains traces of arsenic, a known carcinogen for skin and lungs. He further pointed out that the assessment of risk arising from coal-coke oven exposure is more complicated than the assessment of the risks associated with uranium mining or asbestos manufacture:

> Firstly, unlike the latter two carcinogens, there is little idea even of the quality of exposure in coke oven emissions, and secondly, that, again unlike asbestos or radiation, there is a likelihood that following cessation of exposure to coke oven emissions the risk declines in the same way as in a cigarette smoker, wherein after ten years of abstinence the risk of lung cancer is not much greater than that faced by the non-smoker.

He felt that it would not be justifiable to assign weight to cigarette smoking in the assessment of cause and effect after cessation of exposure to asbestos and radiation since:

> While the risks from smoking would decline, the industrial exposure risk would not, and one could never be sure in the situation where two entirely different co-carcinogens are interrelating, whether the overall risk declines in proportion if one of these carcinogens is removed, i.e. the smoking factor. In the case of the smoking coke oven worker, decline in the risk associated

> with one carcinogen on its removal would logically be expected in the other on its removal, since they share the most prominent carcinogen - benzo(a)pyrene. Finally, we know very little about the absolute levels of coal tar volatiles in coke oven workers, and while this is not too critical in topside workers because of the relatively high risk, it does introduce uncertainty in assessing risk at bench level where exposure is much less hazardous. By including smoking, a more rational approach to cause and effect can be justified.[123]

Thus, although there is some logic to the assumption that smokers would tend to develop their cancers sooner than non-smokers (due to the combined effects of the carcinogens), and there is evidence that the risk of developing cancer from cigarette smoke does eventually drop after cessation of exposure, there is no justification whatsoever for the numbers chosen. That there is a degree of arbitrary judgement involved in the establishment of numerical guidelines is a recurring theme in this chapter. It is specifically an issue in this set of guidelines as the scientific basis for the cessation interval criterion is particularly weak.

Nevertheless, the guidelines were approved in April, 1976 (See page 209). By December of 1975, 17 claims had been received for lung cancer allegedly due to coke oven emissions from employees of a single major steel company. Throughout the year the press had carried reports of opposition members of the Legislature outraged about Government failure to force proper monitoring, of coke oven emissions, especially in the case of one particular Hamilton steel company, [124] and about the fact that trade unions rather than the Province, had to track down workers whose cancer may have been work-related, (notably in another local steel operation [125]. (Interestingly it was noted that although some of the claimants had attended the Cancer Clinic, their occupation was not even reported in medical records).

By the end of 1976 four claims for cancer due to coke oven exposure had been allowed, and by 1978 the Board had accepted eleven lung cancer claims from four companies - three from one company, three from another, four from a third and one from a fourth. Following the corporate takeover of one of the smaller plants by one of the larger ones, records became particularly difficult to track down with respect to precise job classification and duration at risk.

In the Lloyd studies the risk emphasis was placed on the main operating personnel working on top and at the sides of the ovens (bench level), whereas by 1978 the Board was faced with at least two cases of workers involved solely in maintenance. At least one of these cases involved an appeal by the company with respect to exposure. The Board felt that maintenance personnel do indeed spend as much time as the main operating personnel on top or sides of the batteries. Two cases were ultimately allowed involving maintenance personnel (a millwright and a pipefitter both employed by the same large steel company).

Before long it became clear that serious problems existed in defining exposure in the adjudication of claims and these gave rise to frequent disputes between union and management with respect to actual time spent by individuals in precise locations. It was also concluded that past conditions were even worse than present ones, and the Board noted that even in the most recent studies (1976) even the cleanest coke ovens in Ontario had levels of coal tar pitch volatiles averaging always in excess of the threshold limit value of 0.2 milligrams per cubic meter, equal to or slightly higher than in the U.S. The majority were noted, in fact, to exceed 0.54 milligrams per cubic meter.[126] It was thus decided to drop reference to old coke ovens vs. new coke ovens in the adjudication of claims.

On reviewing the literature, the Board noted Redmond's finding that the time from the first exposure to coal tar pitch volatiles to the diagnosis of lung cancer varied from ten to forty years, with the average being twenty-five years. As well, it was noted that at high exposures there was an increase in the mortality rate in the under 35-year old age group, increasing markedly in all subsequent age groups up to and beyond the age of 55. Therefore, the Board's consultant concluded:

> It would seem that an increase in risk is not confined to any age grouping, so we are obliged to consider favourably a claim involving the required exposure with a latent period as short as ten years or as long as forty. If such is the case, the rationale for the use of specific age groupings, either in isolation or in combination with the specific smoking history, as we have incorporated in our present criteria, disappears.

The decision to drop reference to the age factor was thus made. The rationale for inclusion of smoking history criteria was however reiterated on the basis of extrapolating the diminishing risk associated with cessation of smoking to cessation of coke oven exposure.

> If cancer has not developed within the fifteen years after cessation of coke oven risk, then any cancers developing beyond this time in a smoker are not likely related to coke oven exposure. In the case of a proven non-smoker or ex-smoker, lung cancer appearing more than 20 years after exposure has ceased should be presumed to be related to other causes.

The review concluded that ground level jobs were without risk and therefore it was decided to remove this from the guidelines. With respect to relative risk of topside vs. side oven exposure, it was noted that

> The relative risk of topside exposure as opposed to side oven exposure is in the order of three to one. Since there is no reason to alter the five-year minimum exposure for full-time topside work, it would be in order that we continue to require 15 years for side oven

exposure only and ten years for mixed side oven and topside exposure.[127]

Some of these conclusions are controversial and will be commented upon below. On November 17, 1978, the revised set of guidelines was approved (See page 211).

Meanwhile on July 14, 1977, the WCB received a letter from the Director of Occupational and Environmental Health for a major Hamilton steel company. The Director, who was also a member of the faculty at McMaster University, requested that WCB claims be established on behalf of 22 deceased workers who had been employed by the steel company. Each of the 22 workers had 5 or more years of exposure in the foundry and had developed lung cancer. This letter was accompanied by the report of findings of a study which had been completed at the company.[128]

Some abnormalities had been observed in one group of workers in routine respiratory function tests as part of the employer's occupational health monitoring program. Further investigations were undertaken and the Director noted that results indicated that non-smoking foundry workers had respiratory test results similar to non-exposed office workers who smoked cigarettes. The possibility of excess lung cancer in the foundry group was suggested to him simply because it was noted that their pulmonary systems acted "as if they were smokers".

A retrospective follow-up study was designed. All employees and retirees of one steel company who were alive and 45 years of age and over in the year 1967 were identified. This cohort of 1,514 workers was divided into "foundry" or "non-foundry" groups and was followed until January 1st, 1977. The non-foundry group consisted of 1,102 men who had been employed at least five years prior to 1967 but less than five years in the foundry. The foundry group consisted of 437 men who had worked at least five years in the foundry and was sub-divided according to job classification. Eight specific death rates were calculated from 1971 Vital Statistics for Metro Toronto and then applied to the person-years-at-risk for the corresponding employee age group from which total expected deaths in the two groups were calculated. Of the 1,102 non-foundry workers, ten died of lung cancer and of 439 foundry workers, 18 died of lung cancer. (See Table A in Appendix 3c).

There was a significant difference between observed and expected lung cancer deaths for exposures of five years or more, with no significant difference for exposures less than five years. When foundry workers whose initial exposure was more than twenty years prior to 1967 were compared to all foundry workers, there was a statistically significant excess of lung cancer death in those with over 20 years since initial exposure. Smoking histories were lacking, but sampling indicated that no difference in smoking habits between the two groups could be expected.

Air sampling and analysis, however, were unable to identify the causative agent and correlations between levels of exposure and cancer risks were negative as mentioned earlier.

Gibson et al., the authors of the study, stressed that the number of deaths are relatively small and the conclusions drawn from this study must be interpreted accordingly.

The Board's first response to this study was somewhat irate, referring to it as having been precipitiously dumped in their laps. On July 27, 1977, a senior executive in the Board's Medical Services Division, wrote to the company physician who had requested that the 22 lung cancer claims be set up:

> I must state that I am rather distressed in reading the newspaper article from the Hamilton Spectator, in which it is stated that the company has made application for benefits on behalf of the next-of-kin of foundry workers who have died of lung cancer. This certainly would suggest to anyone reading the article that you were convinced that your study was valid and an industrial disease peculiar to and arising out of the employment had been firmly established. At the present time, this is far from being the case... I would like to strongly suggest that if other agencies are going to be involved as the spin-off from such studies, it would be most appropriate to advise these agencies with an adequate amount of lead time. Finally, insofar as the 22 cases on which you have submitted Form 8's are concerned, I must point out to you that at the present time the only criteria for acceptance of such cases would be the criteria which are already established, and which related specifically to coke oven operations. If these 22 cases do not fit into the coke oven operation category they cannot be dealt with under the Act until such time as it is determined whether or not foundry operations do or do not constitute an industrially generated disease factor.... In the interim, we shall hold the data which you have submitted to the Workmen's Compensation Board of Ontario until such time as it is possible to adjudicate these claims from a sound data base.[129]

A copy of the study was promptly transmitted to the newly-formed Occupational Health and Safety Division of the Ministry of Labour for evaluation, search of the world literature and discussion with respect to the necessity of further studies. It was pointed out that "if a decision hinges on this document alone it must be above and beyond scientific reproach". In June 1978, a short memo was received from the Ministry of Labour with a list of references but no further information concerning the status of the study. Meanwhile, the Board had learned of a study being carried out in Quebec at a steel foundry, however, despite many attempts, documentation was unattainable (and is still unavailable).

Guidelines for Adjudication
Lung Cancer - Coke Oven Workers

Recommendations

1. That lung cancer for coke oven workers in the steel industry be accepted as an industrial disease under Section 118 and Section 1 (1) (l) of the Act as peculiar to and characteristic of exposure to coke oven emissions in the steel industry.

2. That based on medical studies lung cancer claims be favourably considered when the following circumstances apply:

   2.1 Persons employed 5 or more years in full time top side jobs.

   2.2 Persons employed as bench level workers in an oven of "old design" and who are in the 40 - 50 year age group and have 10 or more years of exposure.

   2.3 Employees who have been non-smokers for a minimum of 10 years preceding the diagnosis of lung cancer and who were working partially on ground and partially at bench level, with 10 or more years exposure.

   2.4 Employees who have been non-smokers for a minimum of 10 years duration preceding the diagnosis of lung cancer and who are retired coke oven workers and who have worked 20 or more years at ground and bench level.

   2.5 Employees who are smokers or who have stopped smoking in less than 10 years prior to the diagnosis of lung cancer not included in 2.1 or 2.2 and who have worked partially at top side, bench level and ground level for 20 years or more.

3. Claims which do not meet the criteria in "2" will be individually judged on their own merits in view of the variations in exposures which could cause lung cancer and where it seems reasonable that the lung cancer resulted from exposure to coke oven work in the steel industry, and consideration to be given to these cases. The benefit of reasonable doubt applies.

APPROVED BY THE BOARD
April, 1976

On June 28, 1978, a letter was again sent by the WCB to the physician who had requested that 22 lung cancer claims be set up for foundry workers informing him that the Ministry of Labour was still evaluating the study and that in the interim, a Board-employed specialist in respiratory disease cases was continuing on his own to monitor the world literature.

Following several consultations with the Ministry of Labour throughout this period, it was decided that outside services would be sought to evaluate this study. In September 1978, the Board received a reply from the Occupational Health Branch of the Ministry informing that funds had been allocated to McMaster University to study the carcinogenic effects of dust and gases currently encountered in foundry operations of a local steel company.

Meanwhile the long delay had not gone unnoticed. In early November 1978, Hamilton East M.P.P. Bob Mackenzie raised the matter in the Legislature. One of his constituents, a 57-year old foundry worker dying of lung cancer, had made several inquiries to the Board as to the status of his claim. On October 20, 1978, the Board turned him down and according to Stephen Lewis in a newspaper article [130], he had been told that there was not sufficient evidence to establish a relationship between his work and his cancer at that time. Citing the Board's response: "A decision has been rendered at this time at the insistence of yourself and your wife who have called repeatedly, requesting that we proceed without waiting for the results of the studies being carried out."... Lewis was indignant at the Board. It had come to light at this time that "a breakdown in communications" was discovered when a senior Board physician told a reporter that he was still waiting for the results from the Ministry while the Assistant Deputy Minister of the Occupational Health & Safety Division said it was completed. "What makes me so frantic and angry" wrote Lewis lamenting the denial of the benefit of the doubt to the worker and the long delay "is that we have seen exactly the same route before. Stomach cancer at Johns-Manville. Laryngeal cancer at INCO. Now lung cancer at Dofasco." Liberal Leader Stuart Smith echoed his concerns stating that he felt the Workmen's Compensation Board was being "overly careful about linkages of cancer to the workplace that are fairly obvious." NDP Leader Michael Cassidy stated that the families should be paid interim financial compensation, rather than having to wait any longer. Minister of Labour, Dr. Robert Elgie expressed his dissatisfaction at the confusion and promised immediate action.[131]

On November 16, 1978, a senior Board physician approached Dr. A. B. Miller, Director of the Epidemiology Unit at the National Cancer Institute of Canada to develop an in-depth evaluation of the Gibson Study. Dr. Miller's report, which was received by the Board within one month of the request, validated the general conclusion that a risk of lung cancer causally associated with exposure to steel foundry work indeed had been identified in the Ontario plant studied by Dr. Gibson and that the risk did not seem likely to be due to confounding by cigarette smoke.[132]

Guidelines - February 21, 1978
Lung Cancer - Coke Oven Workers

Recommended that:

1. Lung cancer in coke oven workers be accepted as an industrial disease under Section 118 and Section 1 (1) (l) of the Act as peculiar to and characteristic of exposure to coke oven emissions in the steel industry.

2. Based on medical studies, lung cancer claims be favourably considered when the following appropriate combinations of circumstances regarding exposure, latency, and cessation factors apply:

   2.1 Persons employed five or more years in full time topside exposure.

   2.2 Persons employed for ten or more years in mixed side-oven/topside exposure.

   2.3 Persons employed for fifteen or more years in side-oven exposure only.

   2.4 The inception (latent) period between first exposure and the acceptance of lung cancer be at least ten years.

   2.5 The cessation interval (C1) (time between cessation of occupational risk and appearance of lung cancer) in a smoker shall be fifteen years or less.

   2.6 The cessation interval (CI) in a confirmed non-smoker or ex-smoker shall be twenty years or less (see definition below).

3. Claims which do not meet with criteria in (2.) above, be individually judged on their own merits, taking into consideration the variations in intensity and duration of exposure which could cause lung cancer. Where it seems reasonable that lung cancer resulted from exposure to coke oven emissions in the steel industry, consideration be given to these cases. The benefit of reasonable doubt applies.

Definition of Smoking

1. Non-Smoker - A never smoker. A person who has never smoked even occasionally.

2. Ex-Smoker - A person who has smoked during his lifetime but who has not smoked for the past ten or more years.

3. Smoker - A person who currently smokes, or who has ceased smoking for less than the previous ten years.

APPROVED BY THE BOARD
November 17, 1978,

Several of Dr. Miller's comments are well worth noting...

> The data available to me strongly supports the likelihood that a causal association between the work at the Steel Foundry and subsequent lung cancer mortality has been identified... However, it is clear that only extensive and possibly rather expensive additional studies in North America will answer this question precisely, and particularly provide valid data to develop precise guidelines relative to intensity of exposure, duration of exposure, inception period and cessation interval.

He also confirmed that the mortality of foundry workers from all cancers as well as from lung cancer specifically, exceeded the expectation, although only the latter was highly significant. With respect to latent period and duration of exposure, Dr. Miller commented that:

> ... the numbers of individuals exposed in the foundry for less than five years who appear in the non-foundry group are small, nonetheless the apparent lack of increase in risk cannot be accepted without further study of a larger group...Although there is an obvious association between duration and latent period, there is no evidence of clustering of the cases. Thus it may not be supportable (in the legal sense) to place any restrictions on duration of exposure or latent period without further evaluation of a larger experience.

More precise future studies to facilitate further evaluation of the latent period for carcinogenicity and the length of exposure necessary to increase the risk were suggested, including examining the large number of the steel foundry workers not included in the 1967 cohort because they had not accumulated 25 years work experience within the plant. Based on his literature review, he also concluded that an increased risk at other steel and iron foundries in Ontario was also likely and recommended that a study be conducted in all Ontario foundry operations.

The claims submitted for the Hamilton foundry workers were then investigated by the Board's Claims Services Division. On March 12, 1979, the Board physician who was responsible for testing these cases, outlined his findings and observations in detail, based on the then 27 claims before the Board (23 confirmed lung cancers from the foundry studied by Dr. Gibson and his colleagues) and his review of the foundry process. He observed that in the 23 cases submitted the exposure varied from 3 years to 41 years, (mean 21 years), that the latencies varied from ten years to 53 years (mean 31 years), and the cessation intervals varied from 0 years to 25 years (mean 10 years) (See Appendix 3c). His comments included the following:

> The study data are imprecise in respect to risk and its relations to exposure duration and inception period. Despite this observation, increased risk does not appear until about 20 years after the start of exposure and is

> confirmed to those with five or more years of exposure and in the age group 45 to 64 years.

He concluded that (1) a minimum of 20 years latency is indicated, (2) the improvement in ventilation in the 1960's and the spread of cumulative exposure suggested that ten to 15 years of exposure prior to 1956 is critical to the development of occupationally induced lung cancer, and (3) the wide diversity of occupations demonstrated in the cancer cases suggested that increased risk was general throughout the foundry, with risk being higher in certain trades.

In a letter a few months later to the Minister of Labour defending the choice of 1956 as not being arbitrary, he pointed out that the lack of other data meant that the guidelines would have to be solely based on the experience of the Hamilton employer whose workforce had been studied and that (1) a dose-response relationship was unavailable, (2) there was no way of weighting individual exposures and (3) that occupational attribution was clouded by the fact that the majority of claims involved heavy smoking. He explained how the spread in cases suggested that in a majority, latency is independent of exposure duration and since most of the exposure took place prior to the mid-1950's, he concluded that this was the critical period. He explained that improvements in ventilation after the 1950's gave credence to the assumption and that combined with smoking histories, the choice of 1956 was not an arbitrary one. He noted that in 5 of the lung cancer cases of workers whose exposure started after 1950, 3 were probably not industrial as they had short latencies, one worker who was a non-smoker had developed oat-cell cancer representing the reaction of an "unusually sensitive individual" and that the fifth case of a smoker with a long latency of 27 years represented a true smoking cancer. He further pointed out that the minimum of ten years' exposure is substantially less than the average of all the cases, citing this as another reason why a mandatory period of exposure was selected. Finally, he noted that "even with the currently developed guidelines we will end up by assigning as occupational a greater proportion of the cases than would seem to be warranted solely on the basis of the overall standardized mortality ratio as revealed in the Gibson Studies."[133]

The draft guidelines prepared by the Board thus stipulated a minimum 20 year latent period, minimum ten years' exposure within the designated foundry unit prior to 1956 and a maximum cessation interval in a smoker of 15 years and for an ex or non-smoker of twenty years. (See page 236) The Joint Consultative Committee reviewed the report. (At first, concern was expressed within the Joint Consultative Committee Industrial Disease Subsection with respect to the inclusion of the cessation interval criteria. One member stated that he really could not see that the cessation interval had anything to do with cigarette smoking. Upon further discussion, this concern was apparently dropped, unfortunately). The guidelines were approved by the Board on April 10, 1979.

The remaining issue was the question of other foundries. It was agreed that research and investigation should be undertaken to determine whether the lung cancer risk found in the Hamilton foundry studied by Dr. Gibson and his colleagues did indeed exist in other foundries in Ontario. (Some expressed the opinion that this should be the responsibility of the Ministry of Labour, however the Board proceeded to commission the research study.)

It was concluded that a retrospective study of foundry operations in Ontario would take an excessive amount of time, estimated at a minimum of three years, and that it was very probable that the results would be of little assistance in establishing a cause-effect relationship of lung cancer in workers employed in the foundry operations. Consequently, a two-phase program was developed instead. The first phase was a questionnaire to see if there was some common factor in the mould materials or binding materials that were used, as well as the types of metals being poured. The second phase followed this and was a sampling of actual dust and gases in ten typical foundry operations.

The questionnaire was mailed to 199 Ontario foundries, from which 186 replies were received. From this group, 20 representative foundries were visited and nine were selected, along with the foundry that had been studied previously, in which an air sampling program was performed by McMaster University. The results indicated that potential carcinogens were present in all foundries, but that levels varied over several orders of magnitude depending on the type of foundry, ventilation and season. More importantly, it was not possible to identify any characteristics of the emissions of the first foundry studied that were not present in other foundries.[134]

A senior Board physician, felt that in keeping with other carcinogenic situations, a dose-response effect must be assumed and "the apparent weakness of the epidemiological association would support a concept that any risk would only be significant with prolonged high levels of exposure". He felt that the production of PAHs can be anticipated to be minimal in foundries which do not use sand moulds (containing organic binding agents) and that the bulk of exposure to PAHs is presumed to result from direct exposure to fumes produced when metal is poured into the mould, or from exposure to particles released at the time of shake-out or grinding. He observed that ventilation was relatively good where moulds move on an assembly line through pouring and shake-out, although some foundries where pouring occurred at the shop floor did have good ventilation. He, therefore, suggested amending the guidelines based on the study of one particular Hamilton foundry to include other foundries where it can be reasonably deduced that heavy cumulative exposure to PAHs is likely to have occurred based on the above stipulations. He proposed retaining the length of exposure and cessation interval requirements that previously existed in the guidelines.[135] Based on these criteria, guidelines were approved by the Board on August 19, 1981 (See page 237).

3.2.3 *Case Presentations*

As stated, there have been a total of 61 cases allowed so far (December 1981) due to exposure to PAHs. While 16 claims for lung cancer were allowed in coke oven workers, 9 cases of proven primary lung cancer were rejected; 33 cases of lung cancer in foundry workers were allowed while 13 were rejected. In 1980, only a total of 11 new claims were established for lung cancer in foundry workers and only 4 in 1981. As well, only 2 lung cancer claims in coke oven workers were established in 1980 and none in 1981. (One claim for gastro-intestinal cancer in a coke oven worker was also reported in 1980, and 3 G.I. cancer claims in foundry workers were filed in 1981 as well.)

In the Industrial Disease Survey performed for this Inquiry, 10 claims from the 105 reviewed were filed for workers exposed to this group of chemicals: 5 claims were from foundry workers, of which 2 were allowed, and 3 were rejected, (1 rejection was for lung cancer in a worker employed in the operation studied by Dr. Gibson and the other 2 were claims for lung cancer in workers from other foundries); 2 claims were from coke oven workers of which 1 was withdrawn by the claimant as lung cancer was not found to be the correct diagnosis and 1 was an allowed claim for lung cancer in a worker in a steel company's coke oven operation; 1 claim was filed for a worker exposed to cutting oils, 1 from a steel worker exposed to coal-tar emissions, and 1 was from a worker exposed to cigarette smoke at work. Eight of these cases will be presented below.

Case 1 - Claim 18: This 58 year old steel worker died in November 1954. The claim was submitted by the employer in 1979 as it had been found that although no post-mortem had been performed on this worker "possible cancer of the lung" and "possible brain tumour secondary to lung metastases" had been diagnosed. He had worked as a chipper for short periods in 1929, 1930 and 1932 and steadily from 1936 until 1938 and from 1939 until leaving in 1954. The case fell within the guidelines and the claim was allowed.

Commentary: This claim as well as another for lung cancer from a foundry worker, employed from 1928 - 1957, met the guidelines and were consequently promptly allowed.

Case 2 - Claim 19: This steelworker who had worked in the company studied by Dr. Gibson, since 1918, spent 16 years in the foundry and then transferred to the hot mill (not designated as within the foundry area) where he worked until he developed cancer in 1956. The claim was investigated in 1979. A Board-employed physician who reviewed the case concluded that "although it was not known whether he was a smoker or a non-smoker, as the cessation interval was in excess of 20 years (22 years) the claim does not meet the guidelines for allowance of lung cancer even though the exposure of 16 years and a latency of 38 do apply". The claim was rejected.

Commentary: As stated, this case was rejected solely on the basis of failure to meet the cessation interval criterion. In view of the fact that the validity of cessation interval criteria can be challenged scientifically, the rejection of this claim is certainly questionable.

Case 3 - Claim 20: This 51 year old worker developed cancer in 1976. He had worked in several foundries in Ontario from 1948 to 1969 as a moulder and coreman. In October 1978 a Board physician wrote: "As we have no guidelines for consideration of lung cancer claims in foundry workers, I recommend that this claim be rejected."

Commentary: As stated, this case substantiates that it is indeed the Board's practice to reject cases in the absence of very specific proof and particular guidelines. Since the original rejection of this claim, the Ontario Foundry Study has been completed and guidelines have been developed. As this worker had been employed in several foundries, it is uncertain whether or not the claim would now be allowed, as the guidelines are still fairly restrictive.

Case 4 - Claim 21: This 61 year old worker worked for a steel foundry in Welland from 1934 to 1975 as a moulder. He developed lung cancer for which a lobectomy was performed. His specimen showed extensive silicosis and claims for both the silicosis and lung cancer were established. The Advisory Committee on Occupational Chest Diseases felt that although the specimen showed silicosis, there was no silicosis on the x-ray. The Board's consultant pathologist, stated that although there was moderate silicosis of the hilar lymph nodes, there was no evidence of pulmonary silicosis and that the marked focal fibrosis of the lung was probably secondary to the squamous cell cancer. A Board-employed physician who specialized in respiratory disease claims wrote that "(it was) confirmed that there is no silicosis and I do not believe the lung cancer has any connection with his work exposure." The claim was rejected.

Commentary: Little attention was even given to the lung cancer in this individual, presumably due to the lack of guidelines. Again, inadequate industrial hygiene information was accumulated in relation to this case to assess its chances for allowance under the present guidelines. One could argue that even though guidelines had not been established, a greater attempt should have been made to investigate and document workplace exposures. The statement by a Board physician that this cancer is not occupational in the absence of proper investigation is indicative of the Board's often criticized attitude to claims of this nature.

Case 5 - Claim 22: This worker was 50 years old when he died in 1978 of lung cancer of the oat-cell type. He had worked for 15 years topside in a coke oven operation. It was noted that he was a heavy smoker, and had a 15 year latency period. As the guideline requirements were met, the claim was allowed.

Commentary: This case illustrates the straight-forward allowance of a cancer claim by a coke oven worker who clearly met the guidelines. As was Case 1, it is included for contrast purposes.

Case 6 - Claim 23: This 62 year old machinist of 37 years developed oat-cell cancer in 1978. He had worked 25 years since 1954 in cast iron finishing and was exposed to several cutting oils as well as to stainless steel and stellite. According to the foreman, sometimes the metal would get hot and fumes would rise from the machine "like oil smoke". It was noted that he smoked for 35 to 40 years, ceasing five years prior to diagnosis of his cancer. His exposures were investigated by the Occupational Health Branch of the Ministry of Labour. The company manufacturing the cutting oils was contacted for a list of chemicals. According to that petroleum company, all the ingredients were found to be non-toxic and the testing procedures were also documented. The report from the Ministry's physician stated:

> It is well know that cutting oils may cause dermatitis, skin cancer and scrotal cancer. However, there is no clear evidence that pulmonary cancers result from exposure to cutting oils. Also it is well known that dichromates may produce oat-cell cancer. However, I can find no reports of oat-cell cancer arising due to the stainless steel or stellite machine operations.

A Board physician wrote: "I would agree that this man was not exposed to a carcinogenic substance in his employment. It is noted that he did smoke for a considerable number of years. I would agree that this claim should be rejected."

Commentary: In this case an appropriate workplace investigation seems to have been carried out. Although the rigour of the carcinogenicity testing could be challenged (as discussed in Part A of this chapter), the fact that any testing was done is relatively commendable. The statement by a Board consultant that this cancer was not occupational in the absence of thorough investigation, indicates an often criticized approach to claims of this type. It should be noted that, contrary to the Board doctor's statement, the Ministry's report did not state that there was no exposure to carcinogens, only that epidemiological evidence of an increased risk of oat-cell cancer in machine operators exposed to stainless steel or stellite was lacking. Most notable in this case was that the cancer was once again attributed solely to cigarette smoking.

Case 7 - Claim 24: This 50 year old steel worker was diagnosed as having cancer of the larynx in 1976 (although apparently it was suspected for sometime). He worked in a steel-making company from 1953 to 1979 as a general labourer, mouldman, and mould sprayer in the open hearth and steel-making department. In March 1979 a claim was submitted for him and seven others all employed in the steel-making department where they had been exposed to emissions and fumes of coal tar (as well as asbestos dust to some extent) and who all developed cancer of the lung or larynx. It was noted that this worker had smoked about ten cigarettes per day, although the exact duration was not known. It was also noted that he had a history of alcohol ingestion. Nonetheless his own doctor felt that there was a strong possibility that this cancer was related to exposure to the chemicals at work. The Board physician who reviewed the case stated that there was no merit to this claim as it did not meet the requirements under the criteria for laryngeal cancer and in May 1979 the Review Branch confirmed the denial.

An appeal was sought and the union presented evidence confirming that the workers sprayed the inside of ingot moulds with a liquid containing coal tar. They enclosed copies of materials used throughout his 26 years of exposure along with chemical analysis that had been done and reports from the Occupational Health Branch of the Ministry. The Board found that sufficient exposure to asbestos was lacking and that scientific studies did not establish that this worker's exposure to coal tar between 1955 and 1970 had contributed to the development of laryngeal cancer. The appeal was denied.

The union then sought help from the Director of the Occupational Health Program of McMaster University, who thoroughly reviewed the work history and wrote that he believed it would be difficult to envisage an atmosphere more conducive to the development of respiratory tract cancer and that workers who developed cancer of the lung or larynx while in such employment should be accepted as having a work-related disease. Acknowledging that the usual conditions of duration of exposure and lag since the first exposure would have to be considered, he believed that there was sufficient scientific evidence to support the proposal that workers who worked under the conditions described to him were exposed to respiratory tract carcinogens.

A Board-employed physician who specializes in chest disease cases, in considering the letter, pointed out that in the Lloyd-Redmond survey of the steel industry and in Gibson's steel mill study, no increase in laryngeal cancer was noted in any of the steel-making or foundry occupations. He noted that neither Gibson nor Lloyd found increases in laryngeal or lung cancer in steel mill workers, other than in the coke ovens. He also noted that of five workers who developed lung cancer in the group of claims submitted, all were heavy smokers. He felt that the expert physician consulted by the union "relies on the heresay description given by the workers of the conditions that existed in this area of the plant and had failed to note that the working ennvironment described was likely no more and probably very much better than others existing in many areas of the steel, iron and coal gasification industries now and in the past." Again pointing out that no excessive laryngeal cancer had been documented in other industries, he wrote:

> The Medical Branch has never recommended and the Board has never accepted guidelines dealing with occupational cancer that have not been sustained by

> epidemiological studies showing increased risk. A lack of increased risk of lung cancer in respect to sintering in one plant, in the nickel refinery operations (Coniston) as opposed to a high risk of lung cancer at another part of the sintering operation (Coppercliff) illustrates the weakness of assuming that exposure to a substance in one context generates the same risk as exposure in another. This applies equally to exposures involving tars, pitches and asphalts.

(Ironically, more recent evidence suggests that the Coniston operations did indeed share the increased risk observed in other sintering operations, as mentioned in Section 3.3.)

It was pointed out that accepting the thinking of the union's medical expert would involve a major change in philosophy at the Board. It was concluded that "the director's hypothesis must be considered as quite improbable for laryngeal cancer and not even really that probable for lung cancer." The Appeals Adjudicator suggested supplying these memos to the specialist consulted by USWA for counter-argument. Meanwhile a letter was received from the employer (September 1981) expressing agreement with the Board's approach.

Further submissions were received in October 1981 from the union citing studies not mentioned by the Board consultant, for example, where an excess of cancer of the larynx was found in gas stockers and coke oven chargers by Kennaway in 1936, Radford's report of increased cancer throughout the steel industry in 1976, Redmond's study of coke by-product workers in 1975, and a few others. It was also pointed out that the situation in the Ontario steel maker studied by Dr. Gibson was not really comparable since they did not spray the inside of the ingot moulds with coal tar (nor did they use asbestos to line the steel moulds). The union maintained that the emissions and dust from the coal tar spraying operations were unusual and caused the workers to leave the area in which they were working. Letters from USWA Local 1005 Safety Committee to the employer in 1956 and 1954 complaining about "tar smoke" were enclosed, and it was also explained how spraying of ingot moulds with coal tar had finally been stopped because of complaints by the union's safety committee during the 1975 contract negotiations. Meanwhile the Director of the Occupational Health Programme at McMaster had written to Dr. Redmond to ascertain if Redmond and Lloyd looked specifically at mould sprayers. Dr. Redmond's reply stressed that the wording of their survey results was that they were unable to demonstrate a cancer risk and not that a cancer risk was absent. She pointed out that the number of men in their study who were employed in this trade was too small to detect an increased risk (See discussion in Part A of this chapter). To date, the Director has not received the Board memoranda and over three years after the initial filing of the claim, the case has still not been allowed.

Commentary: This case is a classic illustration of the difficulties facing cancer victims in their fight for compensation. This worker (and his seven co-workers) were exposed to a high level of the oldest known carcinogen. Epidemiological evidence has repeatedly linked exactly this type of carcinogen to exactly this type of cancer, however not in exactly the same occupational circumstances. Due to the relatively small number of workers who had been exposed to exactly this set of circumstances, it is unlikely that more convincing epidemiological evidence will ever be produced. Hence the present commitment to vigorous cientific proof of occupational causation will be likely to hinder the provision of justice to these workers. Furthermore it will tend to compensate many worthy claims too late, if at all.

General discussion:

These seven cases show the same pattern of adjudication as occurred in the cancer claims in the previous section, although the issues emphasized were slightly different. The controversy in the claims for radiation-induced cancer focused on the intensity of exposure and the reliability of measurements used. Despite the scientific recognition of the lack of threshold below which no increased risk occurs, claims were often rejected on the basis of insufficient accumulation of exposure. In claims for cancer resulting from exposure to polynuclear aromatics often even the quality of exposure is unknown and measurements of exposure in these situations is not even a consideration in the adjudication process. Instead reliance is placed on years of exposure and, in the situation of coke ovens, job description (topside vs. side oven, etc), and now with the new foundry guidelines, specifics regarding adequacy of ventilation and process used.

Also in this group of claims the variable of cessation interval (time lag between cessation of exposure and diagnosis of cancer) has been added to the criteria for adjudication. The rationale for its inclusion is based largely on the extrapolation of the knowledge from cigarette smoking and some very weak substantiating evidence from the Gibson study. Its inclusion can be questioned on several grounds, including the variables surrounding cancer detection methods, as well as the obvious assumption regarding the extrapolation itself. The issue of cigarette smoking takes on a larger role in this set of claims than in the previous ones, as it plays a role in the cessation interval criteria. It is assumed that in a smoker, the cancer would appear sooner i.e. within 15 years of exposure, whereas 20 years cessation interval is allowed for a non-smoker. Although there may be some logic to the assumption, the numbers are definitely questionable. Case 2 was rejected solely on the basis of not fulfilling the cessation interval criteria although the length and location of exposure as well as the tumour site, type and latency period requirements were all met.

Case 3 and 4 were rejected, it seems, basically because the workers were not from the foundry from which survey results were available. It is anticipated that when the claims are reviewed in light of the completion of the province-wide foundry study, as is the policy of the Board, that the decision in these two cases will be reconsidered and possibly reversed. The effect of the delay on the survivors can well be surmised.

Case 6 illustrates a relatively good industrial hygiene survey by the Ministry of Labour's Occupational Health Branch (in contrast to some discussed in the next chapter). The rejection of this case was based on the lack of evidence of carcinogenicity of some of the chemicals contained in the cutting oils, but also specifically on the lack of epidemiological evidence of increased cancer risk in this particular occupation from exposure to chemicals known to be carcinogenic (e.g. chromates).

Case 7 is a classic example of the controversial nature of the Board's policy which has been described as rigidly conservative. There was no dispute that there was ample scientific evidence to implicate the exposure substance as carcinogenic. (In fact, coal tar is the oldest known carcinogen.) The issue revolved around whether or not evidence existed implicating the particular circumstances of exposure in this claim (and consequently in the other seven cases of cancer in workers on the same job).

As pointed out explicitly in this case, the allowance of cancer claims at the Ontario Workers' Compensation Board is firmly based on the existence of undisputable epidemiological evidence of a cause-effect relationship. It was pointed out in Part A of this chapter that only a small fraction of the chemicals and processes in industry have been tested to any extent (by in vitro testing, animal studies) let alone by epidemiological studies. Further, it was noted that epidemiological studies were subject to a multitude of methodological problems that easily bias the results. The more sophisticated the epidemiological approach, the more expensive and lengthy the undertaking. Consequently very few if any epidemiological studies have been done on which precise guidelines can be solidly based. The control of research funding and priorities, as discussed in Chapter I, may also play a significant role in this regard. Thus the very insistence on high quality epidemiological evidence serves to limit the allowance of claims.

Difficulties in the generalization of the results from one study to another situation have been mentioned. Taking into account the variable working conditions from one plant to the next, exposure patterns are not merely assumed to be parallel. For example, despite the allowance of cancer claims from workers in the Hamilton foundry studied by Dr. Gibson, cancer claims from workers in other foundries were systemically rejected as studies on the specific risks in other foundries had not yet been completed. Even when the same substance is involved in different processes, there is reluctance to generalize the risk found in one process to another. Hence, exposure to coal tar in one occupational setting is not generalizable to occupational cancer in another group of workers exposed to coal tar.

In contrast, although the Ontario Workers' Compensation Board is quite reluctant to generalize positive results when it comes to the acceptance of a claim, the same reluctance does not apply with respect to generalizing "negative" results. (i.e. Case 24 above) The lack of documentation of increased laryngeal cancer in several studies of workers exposed to coal tar was repeatedly mentioned in the denial of claims from steelworkers exposed to coal tar who developed laryngeal cancer, even though there was little to show that their exposure was comparable to those in the studies that failed to show positive results. By contrast the fact that coal tar exposure in cigarette smoke is associated with increased laryngeal cancer was not mentioned, nor were other studies suggesting possible increase of laryngeal cancer associated with coal tar exposure. Thus even when epidemiological evidence exists to link exposure to a certain substance or process with the development of cancer, the selective admissibility of these studies in the individual case is problematic and seems dependent on whether the consideration of that specific study would result in allowance or rejection.

It is certainly valid to consider the many factors that interact to cause cancer - the intensity and length of exposure, the age of the individual and interval between first exposure and appearance of cancer, and so on. No one would argue that these factors are irrelevant. However, it can be argued that the stipulation of numerical guidelines is not based on solid data and therefore, serves merely to arbitrarily restrict the number of allowed claims. The Board argues that these numbers are not arbitrary. However, they certainly are based on much weaker data than the decision to accept the exposure as carcinogenic in the first place.

For example, it was shown that little justification exists for the current coke oven guidelines which stipulate exposure requirements of 5 years topside, 10 years mixed or 15 years side oven. In Table C, Appendix 3b, a significantly increased risk of lung cancer was demonstrated in side-oven workers with as little as 5 years exposure. (In fact, Table F shows that an increased risk was evident when all side-oven workers who had any exposure at all were included.) Moreover, the absence of guidelines (and consequent failure to allow compensation) for cancers other than lung in coke plant workers is quite telling in view of the increased risk some of these workers have for kidney, bladder and gastrointestinal cancer, as shown throughout the tables of Appendix 3b (see especially Table G).

Similarly, there seemed to be no justification for the 20-year exposure requirement for the allowance of lung cancer in foundry workers other than the fact that the majority of claimants for lung cancer from the one Ontario foundry which had been studied, had more than 20 years exposure (see Table E, Appendix 3c). As shown in Table C in Appendix 3c, the study of that particular Ontario foundry demonstrated a statistically significant risk for workers with 5 years or more of exposure, as had been shown in previous studies Moreover, the requirement of 20 years in other foundries as compared to 10 years exposure in the foundry studied by Dr. Gibson prior to 1956 is strictly arbitrary; there is no evidence that even suggests that these figures are appropriate. Again, the 20-year latency period requirement reflects little more than the methodological design of the Gibson study (Table

D, Appendix 3c) and the fact that the majority of claims had a latency period of greater than 20 years (Table E, Appendix 3c). The defense of these guidelines, as articulated by their author, was that they allow "a greater proportion of cases than would seem warranted solely on the basis of the overall standardized mortality ratios." The underlying assumption here is that "average justice" must prevail, even if the restrictions mean that some occupational cancers may well be rejected.

Also, there was no reluctance to extrapolate the cessation interval in cigarette smokers to the occupational experience with PAHs in order to limit the allowability of claims in workers who have been removed from exposure for some years. Yet, this same readiness to extrapolate knowledge with respect to cigarette smoking to other PAH exposures is not applied.

(While on the subject of cigarette smoke, the following case is added with a note of irony:

Case 8, Claim 25: This man worked ten years in a poorly ventilated tavern where the concentration of cigarette, pipe and cigar smoke was described as extreme. The only ventilation was apparently from a door left open in the summer, from which the claimant stated, the dust and exhaust from the unpaved parking lot would blow in. The claimant was said not to be a heavy smoker, smoking perhaps 8 to 9 cigarettes per day and the claimant's family doctor felt that his lung cancer was indeed work-related. The Board consultant wrote: "I really cannot see any possible gain by instituting an investigation in the case of this workplace. We do know that he was a heavy smoker and that approximately 80% of lung cancers are caused by smoking." He concluded that his "personal smoking was the cause, not the employment" and the claim was rejected.

Commentary: Cigarette smoke being such a notorious carcinogen, it is difficult to understand how the Board could feel that an investigation of the extent of workplace exposure was not even merited. The opinion of the family doctor was disregarded here as in many cases to be discussed in the next chapter. Moreover, evidence strongly suggests that "sidestream" smoke may be even more carcinogenic than "mainstream" smoke. In a recent review of the epidemiological as well as laboratory evidence on this subject for Health and Welfare Canada, it was pointed out that "a non-smoker exposed to air heavily contaminated by tobacco smoke inhales as much NDMA (a very potent carcinogen) in one hour as a smoker does by smoking 15 non-filter or up to 35 filter-tipped cigarettes."[136] Thus the adjudication of this case also reflects the view that playing "a contributory role" is insufficient; there must be strong epidemiological evidence that the workplace exposure is the principal factor.

Before turning to the content issues in the adjudication of cancer claims for several other exposure substances, certain problems regarding procedure were illustrated in this section and are worth highlighting at this point. The Gibson study fiasco in which the report sat around for 16 months at the Ministry of Labour while the Workers' Compensation Board apparently was under the impression that an investigation was being conducted, is only the extreme of the situation that exists with regard to delays. Although communication problems can certainly be avoided, the policy of achieving a very high level of scientific certainty before determining the compensability of a claim itself results in substantial delays in the adjudication process. This problem must also be resolved.

3.3 Cancer and Nickel Refining

More claims have been reported and allowed by the Workers' Compensation Board of Ontario for cancers relating to nickel refining than for any other type of cancer, including those relating to asbestos. In fact, cancer in workers employed in nickel refining operations accounted for 232 of the 535 cancer claims or over 43% of all cancer claims allowed by the end of 1981. The operations which were identified to have produced these particular claims in Ontario have long been phased out.

3.3.1 The Scientific Background

Nickel was first reported as a carcinogen of the respiratory tract nearly 50 years ago.[137] This was followed by a study which documented an excess of lung and nasal sinus cancer in workers at the Welsh nickel refinery during the period 1921 to 1932.[138] Subsequently great increases in respiratory tract carcinoma among nickel refinery workers were thoroughly documented in Wales,[139] Norway,[140] Russia[141] and here, in Canada.[142] Thorough review of the research on nickel carcinogenesis led the U.S. National Academy of Science[143] (NAS), the IARC[144] and NIOSH[145] to conclude that an increased incidence of lung cancer and nasal cancer had been clearly demonstrated in epidemiological studies of nickel refinery workers.

There have also been studies suggesting that cancers other than lung and nasal sinus may be attributable to exposures that occur in nickel production. For example, reports of slight increases in kidney cancer[146], cancer of the prostate[147] and stomach cancer [148] have been published, although admittedly they have not produced a consistent picture. The studies regarding the relationship between laryngeal cancer and nickel exposure will be discussed in Section 3.4.

While nickel carcinogenesis in experimental animals has been studied in some detail,[149] the particular causative substances and conditions have not yet been determined. Nickel pellets and dust, nickel subsulfide, nickel acetate, nickel hydroxide, nickel fluoride, nickel oxides and organometallic compounds such as nickel carbonyl, and nickelocenes, have induced cancer in animals after injection, implantation or inhalation. Mutagenicity tests of nickel compounds in some culture systems have shown positive results[150], however, results in bacterial systems have consistently been negative.

By performing biopsies of the nasal mucosa of workers exposed to nickel (as distasteful as these may seem) the gradual transition from normal respiratory membrane to atypia to intra-epithelial cancer to invasive carcinoma has been demonstrated.[151] Some authors have noted that whether nasal, laryngeal or bronchial in location, these tumours were consistently of the squamous cell type,[152] and that the process of carcinogenesis seemed to be the same whether the exposure was predominantly to insoluble nickel compounds found in the roasting-smelting department or due to the largely soluble salts in the aerosols of the electrolytic department.[153] The finding of histological changes in the nasal mucosa of electrolytic workers corresponded with the cancer risk demonstrated in these workers.[154] Mixed exposure to insoluble as well as soluble nickel may have accounted for these findings. However the possibility that soluble nickel (as found in the electrolytic department) is carcinogenic cannot be ruled out. Moreover, there have been reports of cancer in association with exposures to nickel even outside the refining process.[155] These recent studies show that the cancer risk is not limited to the areas previously identified. As Ontario produces much of the world's nickel these issues hold particular relevance in this province.

3.3.2 Evolution of Board Policy

A large nickel refining company established a Canadian refinery at Port Colborne in 1918, initially using cupola furnaces and calcining. A sinter plant started production in 1927. By 1931, the cupola operations had ended and the phasing out of the sintering process took place from 1949 to 1958 or slightly thereafter. A new sinter plant went into operation at Coppercliff near Sudbury in 1948 which operated until February 1963, when it was superseded by the fluid bed roasting process. The sintering process at both plants for producing oxides differed significantly from the process used by the firm to produce a coarse agglomerate of sulfide particles suitable for blast furnace smelting at Coniston. This latter process was also used by another major nickel refining company near Sudbury, and until recently was thought to be without any apparent increase in cancer risk.

By 1958 workers at the sintering plant began noticing that a number of their fellow workers had died of cancer and a suspicion arose that this may have been work-related.[156] J. L. Gagnon of USWA Local 6500, President of what later became known as "The Sintering Plant Action Committee" approached several trade unionists to discuss how to proceed. It was decided to ask that a survey be made by the Ministry of Health.

In 1959 Dr. R. B. Sutherland, then with the Ontario Department of Health, began his first study in the nickel-refining industry at Port Colborne. This study, involving a review of the period 1920 to 1958, was completed in 1960. It confirmed that there was an excess risk of lung and sinus cancer in cupola and sintering operations.[157] He completed a second study in 1968 involving a review of the period 1948 to 1967 at the Coppercliff Smelter, thereby confirming the cancer risk there as well.[158]

In December 1960, a member of the medical staff who specialized in Chest Diseases at the Workmen's Compensation Board wrote a memorandum stating that evidence from the world literature that nickel is an agent responsible for cancer of the nasal sinuses, was overwhelming. He stated that he considered that the evidence produced by Dr. Sutherland conclusively proved that there had been a significant increase in both sinus and lung cancer attributable to exposure in certain employments, pointing out that the risk of sinus cancer death was 30 to 40 times the number expected. He noted that cancers of the sinus and lung are epitheliomatous cancers and possibly the overall incidence in the respiratory tract should be taken, which would produce an increase of more than 10 times the expected - noting that 48% of all cancer deaths at one nickel refinery were respiratory tract cancers. He observed that Dr. Sutherland's results implied that six months' exposure could be adequate to produce respiratory tract cancer in the Port Colborne operations and that no definite latency period should be established. He recommended that the disease could be referred to as a respiratory tract cancer due to nickel refining, and should be dealt with under the general clause covering industrial diseases which became effective on March 31, 1947. He also commented: "It is very unusual and surprising that a single claim has not been filed when such a great number has been affected. It would seem that claims now would never be filed in certain instances unless some action were taken by the Board." Lastly, he commented that he did not believe that many more cases would be produced as it seemed that it had been the early exposure that had produced the greatest effect.[159]

In January 1961, the Board's Chest Disease Committee decided to seek further scientific opinion and inquire from people in England as to their experience before making a definite recommendation to the Board. Meanwhile the claims investigation began.

From the British it was learned that under the National Insurance (Industrial Injuries) Act of Great Britain, both lung and nasal cancer in process workers in nickel refineries are "prescribed diseases", i.e. given the exposure, the presumption is on the side of the worker. Having considered the various implications, it was decided that only certain cases of nasal and lung cancer in refinery workers at the nickel refining plant at Port Colborne should be recognized. Only those employed around the cupola furnaces, the calciners or the sintering machines, but not the anode furnaces, (electrolytic department) should be considered; for nasal cancer one year's exposure must be established, for primary lung cancer three years, and for lung cancer a minimum of five year lag between first exposure and date of disablement would be required. Also, only those who became disabled after March 31, 1947, the date of the provision in the Act, would be eligible. It was noted that this would result in the allowance of 11 of the 12 nasal cancer cases already reported and 13 of the 23 lung cancers. On February 23, 1961, the Board approved these criteria. (See page 231).

On November 30, 1961, in a meeting between executives from a major nickel refining company and the Workers' Compensation Board, the company raised questions concerning exposure to calcining, objected to allowing cases with long latent periods (i.e. the absence of a maximum time lag) and was opposed to the inclusion of the leaching operations in the criteria as suggested by Dr. Sutherland's report. Following the submissions by the company, the Board decided to amend the criteria to require five years' employment for primary lung cancer, where exposure had been in the calciners, instead of the previous three year requirement, and it was agreed that claims involving leaching exposure would be referred to the Board for consideration. (See page 232 for revised guidelines). The company was satisified with these changes.

In January 1969, Dr. Sutherland released his report on cancer at the Coppercliff sinter plant. (see Appendix 4, Table A for selected results) and in April 1969, the Board ordered that the same criteria for Port Colborne be made to apply to sintering operatons at Coppercliff. In July 1971 the Board wrote to Dr. O. H. Warwick of the University of Western Ontario, asking his opinion on the nasal and lung cancer criteria in relationship to the nickel refinery process in Port Colborne and specifically on the etiology of nasal carcinoma in one of the claimants. His observations, received by the Board in October 1971, are worth emphasizing. Warwick noted that since 1957 (the end-date of Dr. Sutherland's Port Colborne Study) an additional 16 cases of nasal cancer had occurred, which not only strengthened the previously observed association with exposure but included three cases in electrolytic workers. He commented that had the original study been undertaken in 1970, rather than in 1959, the increase of nasal cancer in the electrolytic area would have been obvious and the criteria established would almost certainly have recognized this observation. He felt that the eight exposure groups in the study were arbitrarily defined, obscuring the increased risk in certain work exposures such as the electrolytic department. Further, he commented upon the far greater than expected rate of nasal cancers, a relatively rare cancer, "It will be difficult when considering any further cases which may arise to conclude that there is not a causal relationship between the disease and previous exposure anywhere in the general environment of the Port Colborne plant." As an example, Dr. Warwick pointed out that one such claimant who had worked for 36 years in the electrolytic process was certainly exposed to conditions in the general plant environment.

Warwick also took great issue with the statement that the air samples taken in the electrolytic area in 1970 proved to be "so low that they do not suggest exposure to these metals in the electrolytic department is associated with the development of lung or nasal cancer". He pointed out that present air samples cannot be used as evidence that the hazard did not exist 20 or 30 years ago. Further, he noted that a recalculation of the rate of occurrence of primary lung cancer in workers in the electrolytic area would probably indicate that there is an increased risk for lung cancer as well. He felt strongly that the claimant's appeal should be allowed and that the criteria for nasal cancer were too strict and should be broadened to include all those who worked in the general environment of this plant.[160]

An opinion was solicited from Dr. Sutherland who conceded that this claimant as well as another worker who spent 20 1/2 years in the electrolytic department and 2 1/2 months at sintering and calcining and 2 1/2 years in miscellaneous other departments, should be given the benefit of the doubt "on the basis that it is possible that they had sustained the equivalent of one year of exposure at sintering and calcining operations during their early employment".

In 1972, following the retirement of Dr. Sutherland, the then Director of Environmental Health Services of the Department of Health, a nickel company's medical director suggested to the Department and to the Workers' Compensation Board that further revisions should be made to the guidelines. He recommended that the exposure factor for sinus cancer in Port Colborne workers should stay at one year, but that the requirement for lung cancer should be reduced to one year as well. He recommended that the Coppercliff criteria be the same as for Port Colborne for exposure subsequent to 1952, but that in those cases where exposure occurred prior to 1952, that "dirty start-up" should be recognized and the exposure factor be reduced to six months for both lung and sinus cancer.[161] The revised criteria were approved by the Board on November 7, 1972. (See page 234.)

Meanwhile, trade unionists such as John Gagnon of Local 6500 USWA had been continuing to track down affected workers. They had handed out a questionnaire to their fellow workers and visited widows of deceased workers. In March 1973, they called a meeting of all former sintering plant workers and elected a five-person committee to pursue this matter further and to take up compensation claims.

In October 1976, the medical director who had advocated revising the guidelines requested that the Board's minutes dealing with sinus and lung cancer arising from sintering operations be forwarded to his office for examination by the company's legal counsel. The workers had brought their struggle out into the open, specifically lamenting the fact that the Workers' Compensaton Board was using two different criteria in awarding compensation for former sintering plant workers in two Ontario cities. The Board released a statement to the press trying to clarify the confusion. The Steelworkers, however, argued that it was not true that conditions in Coppercliff differed from those in Port Colborne prior to 1952, stating that, in fact, the employer merely moved the same plant and process to Sudbury after it was established in Port Colborne and that conditions in the latter were equally hazardous.[162] On October 28, 1976, the Workers' Compensation Board received a letter from the nickel company agreeing with the Steelworkers' conclusion. No action was taken by the Board in view of the lack of any scientific basis for such action.

Guidelines for the Adjudication of Lung Cancer and Nasal Cancer Claims in Nickel Refinery Workers at Port Colborne

1. That employment around the cupola furnaces, the calciners and the sintering machines (but not the anode furnaces) shall be considered exposure employment.
2. One year of exposure employment must be established to consider nasal sinus carcinoma.
3. Three years of exposure employment must be established to consider lung carcinoma (primary).
4. Where the nasal carcinoma is involved there shall be no minimal time lag between the last exposure and date of disablement.
5. In lung carcinoma, there shall be a minimal five-year time lag between first exposure and date of disablement.
6. In considering both nasal carcinoma and lung carcinoma, there shall be no maximum time lag between first exposure and date of disablement.
7. Any case which appears to be borderline shall be referred to the Board for consideration.

Since the Board Order of February 22, 1961, directing that nasal sinus carcinoma and pulmonary carcinoma at this plant shall be considered an industrial disease under the Act, it must be recognized that reference must be made to the section of the Act which became effective March 31, 1947, and therefore, no case can be considered where disablement occurred prior to that date.

ORDER OF THE BOARD
February 23, 1961.

Guidelines for the Adjudication of Lung Cancer and Nasal Sinus Cancer Claims in Nickel Refinery Workers at Port Colborne - Revised

Nasal sinus carcinoma and lung carcinoma occurring among certain employees of the International Nickel Company of Canada, Limited, Port Colborne, are to be considered under clause (i) subsection (1) of section 1 diseases peculiar to or characteristic of such exposure employment and compensable where the following criteria are met:

(a) Employment around the cupola furnaces, the calciners, and the sintering machines (but not the anode furnaces) is to be considered exposure employment.

(b) One year of exposure employment is to be established to consider nasal sinus carcinoma.

(c) Three years of exposure employment is to be established to consider lung carcinoma (primary), except around the calciners where the exposure employment is to be five years.

(d) Where the nasal carcinoma is involved, there is to be no minimal time lag between the last exposure and date of disablement.

(e) In lung carcinoma, there is to be a minimal five-year time lag between first exposure and date of disablement.

(f) In considering both nasal carcinoma and lung carcinoma, there is to be no maximum time lag between first exposure and date of disablement.

(g) Borderline and doubtful claims and claims which are reasonable in all other respects where there is exposure employment in the leaching operation whether this exposure is alone or in combination with other types of exposure within the plant are to be referred to the Board for consideration.

As clause (i) subsection (1) of section 1 became effective on the 31st day of March, 1947, no disablement which occurred prior to that date is to be considered.

February 22, 1961.
February 23, 1961.
November 30, 1961.

A report produced in March 1977 for the Joint Occupational Health Committee of Steelworkers' Local Union 6500 and a large nickel company, by the company's physician, categorically stated that Coppercliff even in its earliest days was not dustier than its older counterpart at Port Colborne.[163] This report was forwarded to the Board with a request from the Joint Occupational Health Committee that either the Board or the Ministry of Labour conduct an epidemiological study to determine whether there should be a change in the compensation criteria. The newly formed Occupational Health and Safety Division had been identified as the body to evaluate such requests, however, at a meeting in 1978, the Minister of Labour concluded that the Occupational Health and Safety Division could not carry out such evaluations or studies and requested the Board to handle it.

A physician employed by the Board, in conjunction with Dr. Sutherland and Dr. Halliday of the University of Toronto proceeded to establish a protocol for the study. It was found that it was not possible to review the Port Colborne group since the original data were no longer available. Based on a review of the Coppercliff cohort, the authors concluded that there were good reasons to believe that conditions were markedly different after 1952. (See Appendix 4 Table B.) A factor of two was decided upon to weight exposures for the early years in the data analysis. The standardized mortality ratio (SMR) and the standardized incidence ratio (SIR) for seven different exposure group levels were developed. As no expected rates for incidence were available, the authors decided to use a factor of 1.5, although they noted that a ratio between 1.2 and 1.75 could have been equally justifiable.

Data were plotted, a regression line was developed and standard incidence rates for various exposures were estimated. The lack of mathematical precision in this approach was stressed. With respect to Port Colborne, although comparable data were not available, the authors felt that the chronological occurrence of cases did not reveal any sudden increase of incidence as was found to have occurred in the Coppercliff cohort. The authors felt that their analysis confirmed that the conditions prior to 1952 at Coppercliff constituted a higher risk which was approximately double that of the latter period and that it was reasonable to assume that the risk at Port Colborne in any period was equivalent to the risk at Coppercliff for the period 1952 onwards. They acknowledged that analysis of the data to determine the risk for short exposure is equivocal and arguments can be put forward that the data could be interpreted either as confirmation of the current six-month exposure requirement for the Coppercliff start-up or that exposure could be reduced to three months for this period. The extreme potency of the carcinogen involved was again stressed.[164]

On September 8, 1981, the Board approved reducing the exposure requirements to six months for both Port Colborne and Coppercliff with the exception of the high risk period prior to 1952 which was reduced proportionately to three months (see page 239).

Guidelines for the Adjudication of Lung and Nasal Sinus Cancer from Nickel Refinery Workers at Copper Cliff

Carcinoma of the lung and sinus cancer are to be considered as industrial diseases under Section 1(1)(1) of the Workmen's Compensation Act as being peculiar to, and characteristic of, the particular industrial process, trade, or occupation. Where exposure has been proved in the sinter plant at the Copper Cliff smelter, the following criteria shall apply:

(a) One year exposure employment is to be established to consider lung cancer (primary).

(b) One year exposure employment is to be established to consider nasal and sinus cancer.

(c) In all cases arising out of exposure prior to 1952, six months' exposure employment to be established to consider lung and sinus cancer.

(d) In borderline or doubtful claims where exposure may be less than the prescribed six months, the claim may be referred to the Director of Environmental Health Services, Ministry of Health for his opinion.

November 7, 1972

Meanwhile, in March 1981, a letter had been sent by the Joint Occupational Health Committee in the nickel refinery reiterating the previous position that the differences in exposure criteria between Port Colborne and Coppercliff were not justified and that the preliminary report of the retrospective mortality study of nickel refinery workers under the direction of Dr. Muir of McMaster University confirmed this position. The union, of course, wanted the lower requirement to exist across the board. The JOHC also requested a list of all claimants for compensation benefits for respiratory cancer from the nickel company's operations at Coppercliff and Port Colborne along with the status of each claim. After considerable effort of sifting through records kept in different departments of the Board, this list was produced. (It is profiled in Table IIB-21 and will be discussed below.)

Concern was also expressed (notably by Elie Martel, M.P.P. Sudbury East) as to why cancers among former workers at the Coniston sintering plant were not recognized. It was explained to him that the process involved was quite different and that the preliminary results from McMaster's epidemiological study of the employees of another nickel company were indeed confirming the observaton that no increased risk occurred in this process. Ironically, Martel's concern seems in fact to have been quite valid: the final results of McMaster's study of the first nickel refinery have shown that there indeed was an increased risk of lung cancer at the Coniston sintering plant. For those exposed for more than 5 years the risk was increased 5-fold and was highly significant. (P 0.001)[165] (See Appendix 4, Table C) McMaster's study of the other nickel refinery also revealed an increased risk for lung cancer among sintering workers (SMR of 214), but due to the small numbers involved this was not statistically significant.[166] (Statistical significance tests are used to measure the probability (p) of the association occuring by chance. The probability that the greater than 2-fold increased risk found at the second nickel refinery was a chance finding was 12%. As p was greater than 5% the finding was termed "not statistically significant"). Nevertheless, this finding, especially in concert with the Coniston finding is quite different from confirming the absence of an increased risk.

Thus, there has been a fair amount of epidemiological study in Ontario with respect to the cancer hazard of nickel refining. Suffice it to say that many uncertainties still exist and have resulted in guarded policy decisions along the way. The Board has been willing to repeatedly liberalize its guidelines as new evidence emerged, often this liberalization only results after years of frustration and persistent representations from concerned groups. Of interest was the fact that neither medical scientists, like Dr. Warwick, nor the Joint Occupational Health Committee of the nickel company nor the Steelworkers were able to sway the Board from its approach, although it was noted that in earlier years, the company's concerns were indeed reflected in amendments to the guidelines. For better or for worse, no compensation board in any other jurisdiction has been known to be so deeply immersed in scientific research.

Guidelines for Adjudication
Lung Cancer - Foundry Employees of A Hamilton Steel Maker

1. Primary lung cancer in Dofasco foundry employees shall be accepted as an industrial disease under Section 118 and Section 1 (1) (l) of the Act as peculiar to and characteristic of a process, trade or occupation involving exposure to foundry aerosols within the designated foundry unit as shown in the attached plant diagram.

2. Based on medical studies, a review of past foundry conditions, and analysis of claims reported to the Board, cases of primary lung cancer shall be favourably considered when the following appropriate combinations of curcumstances regarding exposure, latency, and cessation factors apply:

   2.1 Ten years minimum exposure within the designated foundry unit prior to 1956.

   2.2 Twenty years minimum Inception (Latent) period between first exposure and diagnosis of primary lung cancer.

   2.3 The Cessation Interval (time between cessation of occupational risk and appearance of lung cancer) in a smoker shall be 15 years or less. (See definition below).

   2.4 The Cessation Interval in an ex-smoker or confirmed non-smoker shall be 20 years or less. (See definition below).

3. Claims which do not meet the criteria in Section 2 shall be individually judged on their own merits taking into consideration the variations in intensity and duration of exposure. The benefit of reasonable doubt applies.

Definition of Smoking

1. Non-Smoker -A never smoker. A person who has never smoked even occasionally.

2. Ex-Smoker -A person who has smoked during his lifetime but who has not smoked for the past ten or more years.

3. Smoker -A person who currently smokes, or who has ceased smoking for less than the previous ten years.

APPROVED BY THE BOARD
April 10th, 1979

Guidelines for Adjudication
Lung Cancer - in Foundry Workers

1. Primary lung cancer in one specified Ontario Foundry and certain other foundry workers shall be accepted as an industrial disease under Section 118 and Section 1 (1) (l) of the Act as peculiar to and characteristic of a process, trade or occupation involving exposure to foundry aerosols within a foundry unit.

2. Based on medical studies, a review of past foundry conditions and analysis of claims reported to the Board, cases of primary lung cancer shall be favourably considered when the following appropriate combinations of circumstances regarding exposure, latency and cessation factors apply:

   2.1 Ten years minimum exposure within a particular designated foundry unit prior to 1956 with 20 years of minimum inception (latent period between the first exposure and diagnosis of primary lung cancer).

   2.2 Twenty years of minimum exposure within any ferrous foundry unit, including the specified foundry studied by Dr. Gibson (except as stated in 2.1), where the following conditions prevail:

      2.2.1 Predominant use of sand moulds.

      2.2.2 "Floor pouring" of castings.

      2.2.3 Evidence that ventilation has been less than satisfactory as determined by a review of records of the Ministry of Labour surveys (for silica) and/or historical data.

   2.3 The cessation interval (time between cessation of occupational risk and appearance of lung cancer) in a smoker shall be 15 years or less. (See definition below).

   2.4 The cessation interval in an ex-smoker or confirmed non-smoker shall be 20 years or less. (See definition below).

3. Claims which do not meet the criteria in Section 2 (including those from nonferrous foundries) shall be individually judged on their merits taking into consideration the variations in intensity and duration of exposure. The benefit of doubt applies.

Definition of Smoking

1. Non-Smoker A never smoker. A person who has never smoked even occasionally.
2. Ex-Smoker A person who has smoked during his lifetime but who has not smoked for the past ten or more years.
3. Smoker A person who currently smokes, or who has ceased smoking for less than the previous ten years.

APPROVED BY THE BOARD
August 19, 1981

3.3.3 *Case Presentations and Discussion*

As of April, 1981, a total of 239 claims for lung and sinus cancer were received from workers in the nickel company's sintering operations. As shown in Table IIB-21a, over 90% of the lung cancer claims received from Coppercliff and 84% of those from Port Colborne were allowed; more than 95% of the sinus cancers were allowed. For the rejected lung cancer claims, as shown in Table IIB-21b, all but one was denied due to insufficient exposure, one other was denied as death occurred prior to 1947. (It was noted earlier that other cancer cases were reported in those who had died prior to 1947, however it must be assumed that these claims were never actually filed). In the two rejected sinus cancers, one was denied due to insufficient exposure and one was denied as the death was due to coronary thrombosis and no disability due to sinus cancer was documented. Several of these claims will probably be allowed upon review, which, according to Board policy, is routinely undertaken when guidelines are revised.

In the Industrial Disease Survey performed for this Inquiry, 25 cancer claims were received from workers exposed to nickel. Of these, 13 were rejected, 11 were allowed and one was withdrawn by the deceased's daughter as lung cancer was not the correct diagnosis. Due to time constraints, and the profile already provided, these 25 cases were not all reviewed as to the diagnosis and details of exposure. Several of the rejected cases seem to have been denied on the basis that exposures were in operations other than sintering, such as Case 1 presented below. In at least one case, the diagnosis of cancer was not confirmed. Several were rejected due to cancer being of the wrong site as in Case 2 below. (Cases pertaining to laryngeal cancer will be presented and considered in the discussion of laryngeal cancer (nickel and asbestos) in Section 3.4.2 below, and account for a few further rejections.)

Case 1 - Claim 26: This worker was diagnosed as having lung cancer in 1979 at the age of 55. He worked from 1950 to 1978 in the Creighton nickel mines primarily as a crusher. It was noted that he was a smoker. As he had no exposure in a smelter or sinter plant, the claim was rejected.

Commentary: This case begs the question of whether exposures in areas other than the smelter or sinter plant are associated with an increased cancer risk. Initial results of the JOHC study have indicated a slight but statistically significant increased risk of lung cancer for Sudbury workers, which was not associated with any particular occupational group. Other studies have also suggested that pockets of increased risk of lung cancer may indeed exist in the nickel industry, even outside the refining process, although consistent evidence is lacking. Further analysis designed to answer this question will apparently be forthcoming.

Guidelines for Adjudication of Claims
for
Lung and Sinus Cancer - Nickel Smelting - Nickel Refineries
Port Colborne and Copper Cliff

1.0 That primary lung and sinus cancer in workers exposed to operations in the calincers, cupola furnaces, and sinter plant at Port Colborne and in the sinter plant at Copper Cliff of the International Nickel Company be accepted under Sections 118 and 1(1)(1) of the Act as peculiar to and characteristic of processes and trades involving such employment.

2.0 Claims shall be favourably considered under the following circumstances:

    2.1 A history of exposure at Port Colborne in the calciners, cupola furnaces, or sinter plant for a minimum of six months.

    2.2 A history of exposure at Copper Cliff in the sinter plant during the years 1952 to 1963 inclusive for a minimum of six months.

    2.3 A history of exposure at Copper Cliff during the years 1948 to 1951 inclusive for a minimum of three months.

    2.4 Claims which do not meet the above criteria shall be individually judged on their own merits having regard to the evidence submitted. The benefit of doubt applies.

APPROVED BY THE BOARD
September 8, 1981

Table IIB-21a

Lung and Sinus Cancers Allowed and Rejected Copper Cliff and Port Colborne

Diagnosis	**Location Allowed**	**Rejected**	**Total**	
Lung Cancer	Copper Cliff	86	9	95
Lung Cancer	Port Colborne	73	14	87
Total Lung Cancer		159	23	182
Sinus Cancer	Copper Cliff	16	0	16
Sinus Cancer	Port Colborne	39	2	41
Total Sinus Cancer		55	2	57
Grand Totals	**214**	**25**	**239**	

Table IIB-21b

Exposure Time of Claims Denied Due to Insufficient Exposure

# of Claims	**Exposure**			
Location & Diagnosis	**Total**	**3 Months**	**3-6 Months**	**6 mth – 1 yr**
Lung Cancer, Copper Cliff Prior to 1952	6	5	1	0
Lung Cancer, Copper Cliff After 1952	3	1	1	1
Lung Cancer, Port Colborne	14	5	6	3 [a]
Sinus Cancer, Copper Cliff	0	0	0	0
Sinus Cancer, Port Colborne	2 [b]	1	0	0

a. One claimant actually had greater than three years exposure, but died prior to 1947.

b. In one claim the exposure was unknown, and the diagnosis was only made at post mortem.

Case 2 - Claim 27: This worker died in July 1959. The diagnosis on his death benefit claim was cancer of the pharynx. He had worked in Port Colborne from 1925 to 1958 for a total of 33 years which included 29 years in the high risk area of calcining. Tissue specimens were supplied to a Board consultant pathologist, from which he diagnosed cancer of the tonsil, stating that this had nothing to do with nickel. Another member of the Board's medical staff reviewed the case and commented that tonsil is classified in the International Classification of Diseases as part of the gastro-intestinal tract; it is a variant of lymphoid tissue and is not covered by the respiratory epithelium. He noted that in all of Ontario in 1975, there were only 22 cases of tonsil cancer and in 1976, 12 cases. In two of the three claims of this type submitted by one nickel company, electrolytic exposure was noted, while in the third, lengthy calcining exposure was recorded. "Clearly this is not a common cancer, but equally clearly, it is not a respiratory cancer." The claim was thus rejected.

Commentary: - Due to the extremely rare nature of this cancer, it is doubtful if an increased risk could easily be detected in most epidemiological studies, for reasons outlined in Part A of this chapter. Thus cancers of this nature may have to be adjudicated on the basis of common sense and benefit of doubt. Interestingly, a statistically significant increase in buccal/pharyngeal cancer was in fact observed in the JOHC study. The authors felt that this was almost certainly due to additional nasal cancers wrongly coded on death certificates, and they identified 7 cases from lists of WCB claims in which they felt this occurred. Quite likely this case was among these 7. No independent pathologist was asked for an opinion in this case.

General Discussion:

The description of the evolution of the Board's policy again illustrates the important role played by Ontario-based epidemiological studies. The major issue of contention in these claims had to do with the specifics of lengths of exposure requirements and specific job descriptions. Despite the inaccuracy of exposure estimates, upon which its guidelines were based and despite numerous requests from concerned parties, the Board was reluctant to amend them. The two cases presented above illustrate two points yet to be fully clarified: in the one, the issue of cancer in nickel miners or other workers outside the sinter plant, and in the other, cancer of sites other than lung or nasal sinuses. The Joint Occupational Health Committee study has already shed further light on these points, suggesting that a slightly increased risk of kidney cancer and prostate cancer as well as lung cancer in non-sinter plant workers may indeed exist.

It should also be noted that a large nickel company has provided $150,000 annually for this study, as was negotiated in the 1975 collective bargaining agreement. The question of responsibility for studies is thus again raised. Moreover, studies such as these can not answer all possible questions and are, by their very nature, on-going. Policy regarding the adjudication of claims in the interim is thus still problematic, especially in view of the negative assumptions adopted by the Board in their absence.

3.4 *Asbestos-Related Cancers*

Asbestos has been the most studied and well-documented of all the carcinogens; the devastating toll taken by this substance has been considered to be one of the greatest tragedies in recent history. Despite the extensive knowledge accumulated concerning the hazards of asbestos, the reality is that (due largely to the long latency period) asbestos-related cancers will continue to plague us for years to come, regardless of the extent of regulation to be enforced. As asbestos is currently the focus of an entire Royal Commission,[167] the state of scientific knowledge concerning asbestos will not be reviewed here. Consequently the following discussion will not adhere to the format used in previous subsections.

3.4.1 General Over-view and Development of Guidelines

As shown in Secton 2, the Workers' Compensation Board received 60 cancer claims in 1981 and 100 cancer claims in the previous year. (There have been approximately 1,000 cancer claims received over the past ten years or so.) By December 1981, the Workers' Compensation Board adjudicated 689 cancer claims based on its guidelines. Of these, 160 or 23% involved asbestos exposure, and 124 of 535 allowed claims, or again 23%, were attributed to asbestos.

Guidelines have been developed for the compensation of asbestos-related lung cancer, mesothelioma, gastro-intestinal cancer and laryngeal cancer. (Details of these guidelines are provided in Appendix 5a.)

The process of developing of these guidelines has been reviewed by Professor Peter Barth[168] for the Royal Commission on Asbestos. His observations are essentially consistent with those made in the previous sections of this report and will be summarized below. (The reader is referred to Barth's study for greater detail.) The sequence of events surrounding the development of guidelines for the adjudication of claims for laryngeal cancer in workers exposed to asbestos will be discussed below in greater depth, as it clearly illustrates some of the major difficulties in the current Worker's Compensation Board policy. Several cases will be presented to illustrate further problem areas identified.

With respect to the lung cancer guidelines, Barth cited the observations of a pathologist on the faculty at the University of Toronto, commissioned by the Board to study the matter:

> Carcinoma of the lung is more likely in those heavily exposed to asbestos, but may occur in those only slightly exposed .... No easy rule is available to determine the duration and intensity of exposure to asbestos needed to make an asbestos worker eligible for compensation if he develops carcinoma of the lung .... All that can be

> suggested at this time is that each case be considered separately and reasonable exposure be accepted.[169]

Barth also noted that while the pathologist consulted by the Board recommended that no minimum exposure rule be adopted, he did recommend that a 15-year minimum latency period rule be applied, adding, however that latitude should be given. A member of the Board's medical staff who specialized in respiratory disease claims indicated his agreement with the pathologist's conclusion that no meaningful formula could be developed employing duration of exposure and fibre count. Barth reported with some surprise that the guidelines ultimately adopted by the Board were considerably more demanding than its own consultants had recommended, stipulating "a clear and adequate history of at least 10 years occupational exposure to asbestos". By contrast he noted that the latency period criterion was established at 10 years, less restrictive than the consultants' views. (It may also be noted that, unlike the guidelines for PAHs discussed in Section 3.2 above, no mention is made of cigarette smoking.) Finally, Barth noted that the consultant pathologist's recommendation that the guidelines specify acceptance of all lung cancers where asbestosis was present was not adopted, although it appeared to be consistent with Board practice nonetheless.

With respect to mesothelioma, Barth noted that the pathologist's recommendations were quite clear: "Mesothelioma can occur in people who have only moderate exposure to asbestos and who show no other evidence of asbestos injury" and "that all with any exposure to asbestos who develop mesothelioma should be compensated." However, again the Board adopted a more restrictive guideline that "there is a clear and adequate history of at least 10 years occupational exposure to asbestos". A 15-year minimum latency period was also stipulated. Barth does point out that the practice of the Workers' Compensation Board is indeed to compensate for mesothelioma wherever an occupational exposure to asbestos can be demonstrated, despite the guidelines (see case 2 below for further commentary.)

The guidelines for gastro-intestinal cancer also began in the work that the Board commissioned from the pathologist it consulted from the University of Toronto. As the link between asbestos and gastro-intestinal cancer was less clear-cut than the situation for lung cancer or mesothelioma, a follow-up study was recommended. Professor A.B. Miller, of the National Cancer Institute Epidemiological Unit was commissioned to review the literature. His report, submitted to the Workers' Compensation Board in 1976,[170] concluded that guidelines be developed linking asbestos to gastro-intestinal cancers of all sites, after a 20-year latency period. Here Barth notes that guidelines adopted were broadly consistent with Miller's recommendations and with those of the consulting pathologist, but were more strict in that they stipulated that the exposure to asbestos "should be of a continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity."

The guidelines for laryngeal cancer are the most recent ones adopted by the Board for asbestos-related cancer. They are discussed in conjunction with the following case, which became a cause celebre in the trade union movement.

3.4.2 *Cancer of the Larynx and Asbestos-Exposure:*

Case 1 - Claim No 29:[171] In 1973 a claim was filed for cancer of the larynx for a worker who had been employed by a nickel company for 26 years. He first noted a sore throat in late 1972, and by May 1973 had undergone extensive neck surgery. Prior to 1968 when he became a maintenance mechanic he had spent 12 years as a mechanic's helper in the iron ore plant. Both here and in the smelter where he worked prior to 1956, he had been exposed to various substances which have been associated with cancer such as asbestos, nickel, cadmium and cobalt. With the help of union documentation he presented the Board with a detailed work history emphasizing the asbestos exposure. The claim was rejected, as was the appeal "on the grounds that there was no scientific basis to establish an association between laryngeal cancer and the substances to which he was exposed."

In 1975 a second appeal was launched. This time the evidence presented by the claimant included supporting documentaton from a Hamilton physician who had reviewed the work history as well as the studies linking asbestos with throat cancer.[172] Meanwhile, the case had attracted attention in the media and on the political front: NDP MPP's, having accumulated further studies on this subject, questioned both Ministry and Workers' Compensation Board officials by letter as well as in the legislature about the decision on this claim.[173] Despite these efforts, the Board again concluded in October 1976 that there was no proven cause and effect relationship between asbestos and laryngeal carcinoma (nor, for that matter, between throat cancer and the other substances to which the worker had been exposed).

During this time more scientific information had become known, notably through the work of the acknowledged world expert on asbestos, Dr. Irving Selikoff.[174] (This study and other studies on the subject are outlined in Appendix 5b.) Dr. Bette Stephenson, Labour Minister at the time, stated that a decision would be taken when Selikoff's findings on laryngeal cancer in asbestos insulation workers had been reviewed. An advance copy of the results of Selikoff's study was thus promptly submitted to the Medical Services Division of the Workers' Compensation Board. Meanwhile, the Board's consultant pathologist, in a report to the Board in 1975[175] on asbestos-related lung cancer had also noted a possible link between laryngeal cancer and asbestos as did his supplemental[176] and final reports in July 1976.[177] In order to confirm the relationship he recommended further investigation. In November 1976, the Medical Services Division announced that the Board had decided to commission Dr. A.B. Miller to conduct a study on throat cancer in Southern Ontario and the Sudbury area.

Dr. Miller proposed conducting a case-control study, using a carefully designed questionnaire to obtain information on occupational and other exposures from patients with newly diagnosed cancer of the larynx. It was agreed that the focus would be on obtaining information not only on asbestos exposure, but also on nickel exposure and Dr. Miller warned that it might take two years to complete the study. The Board agreed to the proposal in February 1977.

The preliminary work was completed and case acquisition and control identification commenced in May 1977. In order to comply with the Board's request that an interim report be submitted by the end of March 1978, the data available to the end of January were analyzed, although they comprised at this time only a total of 62 case-control pairs. Of the 62 cases, 14 had a history of exposure to asbestos compared to 9 controls. (The discordant pair ratio was 13/8 for a risk ratio of 1.6). Eight of the 62 cases gave a history of exposure to nickel compared to 1 of the controls (The discordant pair ratio was 8/1 for a risk ratio of 8.0). Miller noted that on the basis of "ever versus never exposed" the risk ratio for asbestos exposure was non-significant, but significant for nickel exposure. However, 3 of the cases gave a history of exposure to both asbestos and nickel and if these were eliminated he noted that the risk ratio for nickel exposure would become 5 and was non-significant. His report stressed that the results were incomplete and tentative (see Appendix 5c for his conclusions) but he did feel that asbestos was a cause of laryngeal cancer. He also focused on the synergism of nickel and asbestos: "In my opinion there is sufficient evidence to relate cancer of the larynx as an occupationally-related disease, and that in all probability both exposure to asbestos and exposure to nickel are causal factors."[178] He also commented "that there is nothing in our results to suggest that the data of Selikoff are in error."[179]

The Workers' Compensation Board, feeling that public concern dictated the need to accept laryngeal cancer as occupational, proceeded to develop guidelines. No written substantiation existed at all in Miller's report for the intensity and exposure factors proposed in the guidelines, and the latency factor was derived from minimal data at best. (See Appendix 5b for a summary of world-literature on the subject.) The recommended guidelines (see page 247) were approved May 2, 1978 with the clearly stated intention of revising the guidelines "when the case control study has been completed, approximately one year hence."[180]

On May 10, 1978, Workers' Compensation Board Chairman, Michael Starr, announced that the claimant would receive a cheque within a week,[181] but that compensation was being awarded for his exposure to nickel aerosols over 18 years and 9 months, which met the guidelines. His asbestos exposure, the Board decided, was well below the required limit. Although baffled by this rationale, the response to the decision was generally one of rejoicing.[182] Despite the delay, the allowance of this first claim was clearly a breakthrough. (Apparently one throat cancer had been accepted by the Saskatchewan Compensation Board and there had been a number of court cases in the U.S. over this issue).[183] Some warnings, however, did creep into public comments. MPP Floyd Laughren, for example, expressed reservations about the time limits set and urged the Board to be flexible using them.[184] In fact, these guidelines remained operative for over three years, and the statistics seem to give credence to Laughren's concern. To the end of 1981 only 15 claims for laryngeal cancer had been allowed - 10 for nickel exposure and 5 for asbestos, while union officials struggle to receive entitlement for workers who fail to meet the guidelines: 23 claims for laryngeal cancer in workers exposed to asbestos (9) and to nickel (14). (One case of a worker with 14 years and 7 months exposure to asbestos was referred to in Wigmore's review.[185]) (A few cases of this nature which were encountered in the survey of cancer cases conducted for this Inquiry will also be alluded to in the next section).

Dr. Miller's case-control study was finally completed in January 1981.[186] In it he concluded that the study still tended to indicate a relationship between exposure to asbestos and development of laryngeal cancer, but that he was no longer able to confirm his previous opinion concerning a relationship between nickel aerosols and cancer of the larynx. Significant problems were encountered in assessing occupational exposure to both nickel and asbestos and it was not clear that the final resolution was totally satisfactory. Although 204 cases were included in the study, only 13 (and 8 controls) were classified as having asbestos exposure and 13 (and 11 controls) as having nickel exposure. On this basis, the relative risk for exposure to asbestos in males was estimated to be 2.0 (90% confidence limits 0.9 to 4.5) while the relative risk for nickel exposure was estimated to be 0.9 (90% confidence limits 0.4 to 1.9).

Miller noted that although the risk for asbestos was not statistically significant, an elevated risk was compatible with previous studies. He noted that "re-call bias" was not a factor leading to the increased risk, nor was confounding from cigarette smoking or alcohol consumption. His remarks concerning guideline restrictions, cited in full in Appendix 5D, are worth quoting at length:

> The question of duration of exposure to asbestos sufficient to cause disease and latent period from first exposure to disease onset is difficult to examine in the present study because of the small numbers and incomplete information. One case reported intense exposure for one week only. The remainder indicated exposure for at least a year (where duration was recorded). As far as latent period is concerned, only one case and one control reported a latent period of less than 10 years. Ten years or more from first exposure would therefore seem to be an appropriate division.

With respect to nickel he noted the following:

Guidelines for Adjudication of Claims for Laryngeal Cancer in Industry Related to Asbestos Exposure and Nickel Exposure

Recommendation

1. That laryngeal cancer in workers occupationally exposed to asbestos fibre and/or to nickel aerosol in specific industrial processes be accepted as an industrial disease under Section 118 and Section 1 (1) (l) of the Act as peculiar to and characteristic of such processes.

2. That based on medical studies, claims for laryngeal cancer be favourably considered under the following circumstances:

   2.1 Any industrial process in the nickel industry which produces nickel in aerosol dispersion whether in combined or elemental form. This may include the following:

   Roasting
   Smelting
   Refining
   Welding
   Electroplating

   2.2 Any occupation in which there is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity.

3. **Duration of Exposure**

   3.1 Nickel - an accumulative minimum of 15 years exposure to nickel aerosols as defined in 2.1.

   3.2 Asbestos - an accumulative minimum of 10 years proven exposure as defined under 2.2.

   3.3 Nickel and Asbestos - an accumulative minimum of 7.5 years nickel as well as 5 years asbestos exposures in the case of dual exposure.

4. **Inception Period**

   4.1 Nickel - this shall be a minimum of 20 years from the commencement of the first hazardous exposure.

   4.2 Asbestos - this shall be a minimum of 20 years from the commencement of the first hazardous exposure.

   4.3 Nickel and Asbestos - this shall be a minimum of 15 years from the commencement of the first hazardous exposure.

5. Claims which do not meet the guidelines should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. The benefit of reasonable doubt applies.

May 2, 1978

Guidelines for Adjudication of Claims for Laryngeal Cancer in Industry Related to Asbestos Exposure

Recommendation:

1. That laryngeal cancer in workers occupationally exposed to asbestos fibre be accepted as an industrial disease under Section 118 and Section 1 (1) (l) of the Act as peculiar to and characteristic of such processes.

2. That based on medical studies, claims for laryngeal cancer be favourably considered under the following circumstances:

   2.1 Any occupation in which there is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity.

   2.2 Duration of exposure - an accumulative minimum of 10 years proven exposure as defined under 2.1.

   2.3 Inception period - a minimum of 20 years from the commencement of the first hazardous exposure.

3. Claims which do not meet the guidelines should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. The benefit of reasonable doubt applies.

May 5, 1981

> The lack of increased risk for exposure to nickel is a change from the results of the preliminary analysis of the first 62 case-control pairs. This is an indication of the dangers of drawing conclusions from relatively small numbers of subjects. However, the number of subjects with a history of exposure to nickel was small, and therefore the study does not provide strong evidence for the absence of an effect.

Several senior members of the Board's medical staff analyzed Dr. Miller's report and came to different conclusions:[187] "... both cigarette and alcohol consumption were found to have a strong association with laryngeal cancer. Only 3 of the matched pairs were non-smokers and it is deduced that the risk for non-smokers was exceedingly small ...". In reference to the literature review they wrote: "...Review of other occupational exposures indicated the possibility that metal processing, driving vehicle fumes, foundry fumes, and metal dusts might have relative risks for lung cancer greater than that apparent for asbestos". With respect to nickel they concluded that Dr. Miller's prior conclusions and recommendations concerning nickel " ... can now be discounted ... and hence the guidelines should be amended to delete all reference to nickel exposures." With respect to Dr. Miller's conclusion that a 10-year figure for latency would be appropriate they concluded that "it is apparent that there is quite inadequate new evidence upon which one could base recommendations for changing the minimum latency from 20 years - particularly in view of the discretionary variable permitted for individual claims in all guidelines." On May 22, 1981, the Board's Medical Services Division recommended that the Board approve the amendments to the guidelines with respect to cancer of the larynx, as proposed.[188]

It is quite revealing that the Board's medical staff felt that the evidence to drop recognition of nickel-related laryngeal cancer was adequate, but that evidence to lower the latency period to 10 years was inadequate. In fact, this entire sequence of events is characteristic of the reasons for the controversy over Board policy. The Board had the opportunity to accept the link between asbestos and laryngeal cancer in 1976 when its consultant pathologist made his final report. A second chance presented itself when Selikoff's data became available but was also rejected. The decision to formulate guidelines in 1978 after Miller's interim report was clearly political in nature. Miller's report was admittedly scanty and really added no new evidence on the scientific front.

Moreover, the proposal to rescind the recognition of nickel-related laryngeal cancer is not well supported by either of two recent studies[189,190] in the nickel industry which confirmed the findings of earlier reports that a slight risk of laryngeal cancer exists in the nickel industry.[191] In one study a statistically significant increased risk existed for all exposures to nickel and was particularly high among miners and mill workers; the JOHC study suggested a slight overall excess in the smelter area with a particularly high risk in a select group. The apparent inconsistencies require further study. On last review the WCB was awaiting comments from the parties involved before proceeding.

3.4.3 Case Presentations and Decisions

(The following cases were selected from reviewing a sample of claims for this Inquiry. They therefore are not necessarily the best possible illustrations of the points to be made).

Case 2 - Claim 29: This worker died of lung cancer in August 1979 at the age of 37. He was a non-smoker but had worked from 1965 to 1978 in drywall and insulation work entailing exposure to asbestos. He had worked for numerous different companies, but always in the same trade of drywall and insulation installation. It was extremely difficult to provide the exact dates that he worked for specific companies or even the names of the companies, as in this trade, apparently a job may last for a few days, a few weeks, or a few months. However, the basic job description and the materials handled differed little. The actual sheets of drywall contained a certain small percentage of asbestos fibres and during the sanding operation there would be free dust in the air. It was noted that the claimant frequently worked in small rooms and confined areas, and that he never wore a mask or protective clothing. In addition to the drywall insulation it was noted that the claimant had done some installation of insulation produced by a major asbestos manufacturer, but it was noted that insulation installation accounted for less than 40% of his time.

The investigator felt that it would not be useful to contact the few employers named in the file, as this would be unlikely to add to the basic issues at hand. The Board consultant noted:

> His exposure to asbestos fibres was at most 13 years prior to the development of his lung cancer, whereas most asbestos-related lung cancers were first exposed at least 20 years prior to the development of the cancer. In addition, during those 13 years the exposure was relatively light, intermittent, and did not result in other asbestos effects on lung parenchyma or pleura.

Ironically, it was also noted that: "The patient was basically a non-smoker and lung cancers related to asbestos exposure have invariably occurred in workers who have an established smoking habit or have been ex-smokers with a past history of heavy smoking."

A Board physician who specializes in respiratory diseases, reviewed the case and referred to the fact that the family physician and attending consultant also indicated doubts regarding work-relatedness. Summarizing the case he wrote:

> All in all this exposure was intermittent and light and his cancer appeared at a relatively young age. Under ordinary circumstances and in those whose exposure is generally of a continuous and moderate to severe nature, the average latency period is between 20 and 30 years. In this claim the cancer appeared 12 years after exposure started. You might expect such a reduction in the latent period in a heavy smoker and in one who was also heavily exposed to asbestos but neither was the case here.

The case was thus rejected. An appeal was launched and documents from noted experts such as Nicholson and Selikoff were introduced to show that drywall construction is associated with considerable risk of exposure to asbestos. (The appeal was still pending at last review.)

Commentary: As with cases presented in previous sections, this case again illustrates the difficulties that face workers in proving the actual extent of their exposure to the carcinogen in question. The fact that the worker sustained asbestos exposure throughout his 13 year work history was not in question, only the level of the exposure was doubted. As numerous small companies were involved, proof of extent of exposure was completely unattainable and clearly "the benefit of the doubt" was not awarded on this point.

The failure to meet the 20-year guideline (required for cancer to develop in "most" workers), and the failure to meet the "average" latency period were cited as well. As the definition implies that half the workers would fail to meet the "average" figure this argument is untenable. The lack of asbestosis, also referred to, should not have been mentioned as it is well-established that asbestos-related cancers can occur in the absence of asbestosis.

Lastly, the reference to the smoking history, or specifically, the fact that he was a non-smoker, epitomizes the "damned if you do-damned if you don't" situation facing the worker. As illustrated in other sections, when a lung cancer claim is rejected in a worker who is a smoker, the cancer is almost always attributed to the smoking. Here, instead of strengthening the likelihood of occupational attributability, the absence of smoking was used to weaken the case. Admittedly the logic itself is not faulty, but the selective use of this rationale limited to cases where it hinders the allowance of the claim, is indeed questionable. Moreover, the statement that asbestos-related lung cancer invariably occurs in smokers has now been shown to be fallacious.

Case 3 - Claim 30: This worker was diagnosed as having mesothelioma in 1974 and died 2 months later. He had worked for Imperial Oil from 1915 to 1961, during which he apparently spent 6 years as a boiler-maker's helper. As insufficient exposure to asbestos was demonstrated the claim was rejected.

Commentary: Even though mesothelioma is a hallmark cancer of asbestos exposure with few cases reported in the absence of documented asbestos exposure, the Board's policy is to deny claims if occupational exposure cannot be proven. In fact, official Board statistics indicate that 6 claims for mesothelioma in the presence of asbestos exposure have been rejected, and 51 allowed, in the Board's history.

Case 4 - Claim 31: This worker had been a boiler maker for 35 years in which it was stated that he "wrapped asbestos around his torch in order to make the flames shoot further". He, too, had worked for numerous employers and it was noted that "there is nothing on file to show that asbestos was used in the major part of this man's work." There was some question as to whether the diagnosis was mesothelioma or (adenocarcinoma) primary lung cancer. His medical records indicated that his pleural effusion may have been due to mesothelioma. The Board's pathologist wrote: "The appearance of the tumour is not sufficiently distinctive to permit a definite opinion as to its origin. The tumour could be a mesothelioma but equally the tumour could be an adenocarcinoma, primary in the lung or elsewhere ..." The claim was rejected.

Commentary: In this case, as with case 2, the work history was impossible to trace. The uncertainty with respect to tumour type seemed to be a large factor in the rejection. Perhaps had mesothelioma been confirmed the claim would have been allowed.

Case 5 - Claim 32: This worker died in August 1978 of carcinomatosis (or widespread cancer). He had been an asbestos installation worker for about 30 years, and had worked for a number of companies. An autopsy was carried out but the nature of the primary tumour was not easily identified. The independent pathologist's report stated that it looked like mesothelioma, but the report of the Board's consultant pathologist in January 1979 concluded that the primary was cancer of the pancreas. The claim was rejected and in the appeal a brief was submitted by the union representative stressing the 30-year exposure to asbestos and that even if the diagnosis was cancer of the pancreas, there was a biological possibility that this type of cancer also developed from exposure to asbestos. It was also noted that the description of the Board's pathologist was highly tentative as the autopsy material had been poorly preserved.

The union representative suggested that the worker's symptoms prior to death were more consistent with mesothelioma than cancer of the pancreas. He suggested that a separate panel of doctors be setup to examine this aspect. The appeal board decided to submit the matter to a senior member of the Board's medical staff who, in turn, suggested that the original pathologist's slides and report be sent to the Board's consultant pathologist and his report be considered final. (On last review, December 1981, the report was still pending.)

Commentary: Again, determining the original tumour site was problematic (See Section 2.1 of Part A of this chapter for explanation.) The exposure history in this case was certainly of adequate duration. This case illustrates a Board tendency to prefer the opinion of its consultants over independent medical opinion.

Case 6 - Claim 33: This worker had sustained a high level of asbestos exposure from the notorious conditions at an Ottawa gas mask plant where he worked from 1939 to 1945. Slides obtained at autopsy were sent to the Board's consultant pathologist who concluded that the diagnosis in this case was metastatic cancer perhaps originating in the pancreas, colon or stomach. The Board consultant noted that "in view of his short exposure to asbestos fibres ... and since this man does not have mesothelioma, he has not sufficient exposure to consider allowance of this claim for gastro-intestinal cancer in an asbestos worker." The claim was rejected.

Commentary: This case is similar to the last one in that the primary cancer could not be diagnosed. Here it is not the intensity but the duration of exposure which is ultimately responsible for the rejection.

Case 7 - Claim 34: This claim for cancer of the larynx came from a worker who had been exposed to asbestos as a furnace man from March 1969 to May 1977. It was established that asbestos blocks were used to wipe the wire after it came out of the flux, but it was noted that the claimant worked in close proximity to the asbestos blocks only intermittently. As only 8 years and 3 months of proven exposure had been accumulated, it was noted that he failed to meet the requirement of 10 years of proven exposure to asbestos. It was also felt that his exposure was not of a continuous and repetitive nature. The claim was therefore denied.

Commentary: This case was presented simply to illustrate the restrictive nature of the arbitrarily decided upon "10 year view of his short exposure to asbestos fibres ... and since this man does not have mesothelioma, he has not sufficient exposure to consider allowance of this claim for gastro-intestinal cancer in an asbestos worker." The claim was rejected.

Commentary: This case is similar to the last one in that the primary cancer could not be diagnosed. Here it is not the intensity but the duration of exposure which is ultimately responsible for the rejection.

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Commentary: This case was presented simply to illustrate the restrictive nature of the arbitrarily decided upon "10 years of proven exposure" guideline.

Case 8 - Claim 35: This 54 year old submitted a claim for cancer of the rectum (gastro-intestinal cancer). He had asbestosis and marked pleural fibrosis from his exposure to asbestos from 1951 to 1978. It was noted that he had multiple polyposis, which is a familial disease of the colon and rectum. A Board doctor who reviewed the case commented that 30-80% of individuals with this condition develop cancer and therefore concluded the following: "In face of sufficient asbestos exposure we have a non-industrial condition in which the odds of developing cancer are very high and one need not postulate asbestos exposure to account for the rectal cancer." The cancer claim was denied.

Commentary: This case was included to illustrate that meeting all the criteria in the guidelines does not result in automatic allowance of a claim. Here extenuating circumstances, i.e. the familial predisposition, resulted in rejection despite the fulfillment of all the stipulations for gastro-intestinal cancer in an asbestos worker.

General Discussion:

The pattern of adjudication of asbestos-related cancers is not unlike the pattern described for the adjudication of other cancer claims. Despite the extensive study of asbestos, the lack of firm scientific basis for the guidelines remains problematic. The adoption of guidelines more restrictive than those proposed by the consultants commissioned to study the matter does indeed seem to restrict the allowance of claims. Moreover "average" latent periods or exposure durations were referred to in the development of guidelines, with little reference to standard deviations. The standard deviation describes how widely dispersed the values are around the mean, or average. For example, the rationale for requiring a 20-year latency period in laryngeal cancer claims was derived from the findings that the latency period in Selikoff's study "was mostly 25 years or more", the latency period in Still and McGill's study "averaged 30 years" and in Miller's cases "most of the latent periods were more than 30 years and none less than 16 years". It was not noted that the range of latency periods in Still and McGill's study was 3 - 54 years; with a standard deviation of 12 years. Shettingara and Morgan found that the latency period ranged from 5 to 55 years. (See Appendix 5b for description of these studies). Thus the guideline of a 20 year latency period would fail to include claims that are within 1 standard deviation from the mean, let alone the more customary reference point of two standard deviations. Although some of these cases may not have been due to the occupational exposure studied there is little scientific justification for denial of claims based on this guideline.

Cases were presented that reveal the difficulties facing workers in proving their exposure. The problems related to correctly diagnosing the primary tumour type were alluded to, as was the "in-house" nature of the decision-making. The double-standard with respect to smoking histories was pointed out, as was the double-standard with respect to scientific rigour in development of guidelines. The policy seems to be always to err on the side of "accepting the null hypothesis", i.e. assuming no occupational relationship exists until conclusively proven otherwise.

Interestingly, Case 8 disproved the commonly-held belief that compliance with the guidelines results in automatic acceptance, although, admittedly this was the only exception found in the claims surveyed. Finally, the procedure for development of guidelines was again shown to be intensively influenced by events on the political front.

While these factors are certainly problematic, Barth has concluded that Ontario still does well in comparison with other jurisdictions. From 1970 to February 1981 the British Columbia Worker's Compensation Board had apparently allowed only 7 of the 12 asbestos-related lung cancer claims filed (i.e. 58% compared to Ontario's record of 84% allowance - see Table IIB-7). All fourteen claims for mesothelioma filed in B.C. were allowed,[192] (i.e. 100% compared to only 89% in Ontario - see Table IIB-7).

The Royal Commission on Asbestos will undoubtedly address the problems identified above with respect to compensating asbestos-related cancers; many eyes are upon the Ontario Worker's Compensation Board and a new trend begun here may have far-reaching effects well beyond this jurisdiction.

3.5 *Cancer Caused by Other Substances*

Aside from the exposures already discussed, guidelines exist for allowance of claims for one other substance recognized by the Board as carcinogenic, that is, arsenic. Guidelines have existed for the adjudication of lung cancer claims in workers exposed to arsenic since 1956 and since then 33 have been allowed and 12 rejected.

As stated earlier, the only claims to have been allowed by the Board other than those for which guidelines apply are for skin cancer in workers exposed to coal tar, pitch, paraffin, oil and related substances, as prescribed in Schedule 3 of the Act. As well, five claims for bladder cancer in workers exposed to aniline dye were allowed and four cases of leukemia, three attributed to radiation exposure and one to benzene. One claim for synovial sarcoma has also been allowed. (See Table IIB-7). In all these cases, the scientific evidence, specifically the epidemiological evidence, linking these particular cancers to the exposure in question is overwhelming and beyond dispute. The numbers of rejected claims for either bladder cancer or leukemia are not readily available, and none of these fell within the random sample of cases included in the Industrial Disease Survey carried out for this Inquiry. Claims from cancer victims whose carcinogenic exposure does not fall into one of the categories of radiation, PAHs, nickel or asbestos, are filed nevertheless. As noted in Table IIB-9, these accounted for at least one quarter of the cancer claims sampled by this Inquiry. (As all cases were not personally reviewed, others in which the exposure may have been classified in one of the above categories may more correctly belong to this group. Needless to say these categories are gross over-simplifications, as often exposure is to a mixture of carcinogens.) Three short cases are presented below for discussion.

Case 1 - Claim 36: This 40 year old worker developed lung cancer in 1978. He had worked for 25 years at a toothpaste and cleanser manufacturing company, including 18 years in the detergents department. His family doctor filed the claim as he was aware that his patient was being monitored by the Occupational Health Service of the Ministry of Labour and hence, the connection to the workplace was made. At the request of the WCB, the Occupational Health Branch investigated the chemicals in use at the plant. It was also noted that the claimant smoked 1 1/2 packs of cigarettes a day. A Board-employed physician who reviewed the case wrote: "His claim has no merit as this man has not been exposed to any of the carcinogens that are recognized by the Workmen's Compensation Board as being a causative factor in the development of bronchogenic carcinoma." The claim was rejected.

Commentary: The operative phrase in this decision is "carcinogens that are recognized by the WCB". The process by which carcinogens become "recognized" is clearly the issue.

Case 2 - Claim 37: This was a claim for a 62 year old worker who developed lung cancer. He had been a painter for 20 years and also did some auto-body work. The claim was referred to the Ministry of Labour for investigation of the solvents and paints involved. The Board noted that the claimant had been a heavy smoker, and based on the results of the investigation, the case was rejected.

Commentary: Little information is given in the file as to the nature or extent of exposure, although it is well-known that there are many carcinogens to which a worker in this occupation may be exposed. (Painters may be exposed to the potent carcinogen, zinc chromate, for example.)

Case 3 - Claim 38: This 38 year old man developed oat cell cancer. He was employed by a rubber company from 1968 to 1970 where he was exposed to a great number of chemicals. His employment was investigated. A member of the Board's medical staff who reviewed the case had instructed the investigator as follows: "We want a complete smoking history of this man, i.e. the age at which he started, the number of cigarettes per day. If he smoked, we want to know the date . . . whether he smoked plain or filter." An investigation of the chemicals to which he was exposed was performed by the Occupational Health Branch of the Ministry of Labour. The Industrial Hygiene Report revealed that from 1967 to 1970 his work involved calibration of pouring leads on a production line involving exposure to small quantities of many different chemicals. The chemicals were reviewed and none were felt to be carcinogenic. It was also noted that the period of exposure was rather short and the latency rather brief for most lung cancers. Although it was noted that he only had an eleven-year smoking history, smoked only five to ten cigarettes per day and was relatively young to have lung cancer due to smoking, it was felt that it was unlikely that his claim had merit and the claim was denied.

Commentary: As with the previous two claims, this case illustrates the manner in which the smoking history can be diversionary. Ironically, here it was felt that smoking was unlikely to be able to explain the development of cancer. Nonetheless, occupational attributability was not invoked either.

General Discussion:

Without thoroughly reviewing the literature and the work histories it is difficult to evaluate just how much merit each of these cases has. In all of the cases, there was at least some exposure to at least one chemical process listed by NIOSH and/or the IARC as possibly or probably carcinogenic. (Refer to the Appendices of Part A of this chapter.) Although industrial hygiene assessments were indeed undertaken by the Ministry of Labour in conjunction with these claims, with the long latency periods involved the quality and quantity of exposure may have changed drastically and would be difficult to ascertain with any degree of precision under the best of circumstances (as described in Part A). Furthermore, carcinogenic testing of chemicals currently in the work place is grossly inadequate, although some attempt is now being made to screen future chemicals. The difficulties involved in appraising carcinogenic exposures have been discussed above.

It is noteworthy that a great deal of emphasis is placed on obtaining the details of the individuals' smoking histories, as if trying to find an easy explanation. It is interesting, though, that even when the smoking history fails to be able to explain the cancer, the likelihood of occupational attribution does not seem to be increased.

These cases serve to highlight the difficulties involved in adjudicating cancer claims. They strongly suggest that some form of scheduling recognized occupational carcinogens or circumstances of increased cancer risk is necessary to facilitate this process. Perhaps better use could be made of the IARC or NIOSH listings.

4. The Issues Summarized

1. Although cancer claims make up only a tiny proportion of current claims handled by the WCB, they constitute a significant percentage (almost 20%) of the fatal claims reported, even at present. Cancer claims have actually received considerable attention at the Board; in fact, Ontario has clearly played a leading role in conducting research and developing policy with respect to occupational cancer. Three approaches have been used in Ontario: scheduling the particular cancer as a disease under the Workers' Compensation Act, establishing guidelines for claims adjudication, and adjudicating each case on its own merit.

2. Nonetheless, even by the most conservative estimates the number of cancer claims being reported to the WCB falls far short of the calculated number in Ontario. As was shown in Table IIB-15, less than 15% of estimated occupational cancers are reported. Interestingly, this view is in conflict with the position taken by the medical staff of the WCB; in a recent publication they conclude that the Board's experience does indeed reflect the extent of occupational cancer in the province, based on the estimate that less than 1% of cancers are occupationally-related. Relying upon records of upheld compensation claims to document the extent of occupational disease is certainly questionable. For reasons noted above, it produces figures which fall far short of even the most conservative scientific estimates. More importantly, this conservative outlook may account for the rather weak initiatives taken by the Board to seek out workers or their survivors who have a right to compensation.

3. It is noteworthy that the percentage of cancer claims rejected by the Board is almost thirty times higher than the rejection rate of claims generally. Calculations based on data from three different sources within the Board all show that over 50% of the cancer cases reported are rejected. (Tables IIB-3, IIB-4, IIB-5). However, when considering only those claims which are addressed by guidelines only about 22% are rejected (Table IIB-7 upper left). This observation and the fact that only ten cancer claims have been allowed in the history of the Board which are not governed by guidelines underlines the importance placed upon the existence of guidelines. (This excludes skin cancers, scheduled under the Act.)

4. The decision to formulate guidelines is based upon the existence of rigorous epidemiological evidence linking the specific occupational exposure in question with the specific cancer. Evidence of carcinogenicity from animal studies does not constitute adequate evidence, nor is there extrapolation from one set of occupational circumstances to another (as was illustrated by the recognition of cancer from one foundry but not from others until recently, or the recognition of cancer from the nickel refineries at Copper Cliff and Port Colborne, but not from Coniston or elsewhere, for example). The one noted deviation from this rigorous approach was the initial formulation in 1978 of guidelines for the allowance of laryngeal cancer in asbestos and nickel-exposed workers. The political pressure at this time was clearly the reason for this atypical haste. (Interestingly, although this "haste" was atypical, recent evidence suggests that the action may not have been so hasty after all. Studies in both nickel companies did indeed suggest that a slightly increased risk of laryngeal cancer may exist in nickel workers. Ironically, just months prior to the completion of these studies, the Board proposed to rescind the guidelines for allowing laryngeal cancer in nickel workers, in an attempt to rectify what it perceived as a previous error on its part).

   However, this rigorous approach has meant that not all substances for which evidence of carcinogenicity exists have been acknowledged by the development of guidelines. No compensation has ever been awarded for exposure to many internationally-recognized occupational carcinogens (see Part A of this chapter, especially Appendices 3 and 4). In fact, it seems that only where public pressure has dictated the need for recognition, have steps been taken by the WCB to formulate guidelines. Although ample evidence exists, for example, that exposure to chromates causes a markedly increased risk of lung cancer, no guidelines exist in this regard and no cases have been recognized. There is also overwhelming evidence of an increased risk of bladder cancer in certain occupations including rubber workers and leather workers, yet again no guidelines exist in this regard and only 5 cases of bladder cancer have been compensated in the history of the Ontario WCB. The lack of guidelines relating to compensability of leukemia is also noticeable in view of the strong consensus in the scientific community that an increased risk of this disease occurs in the petrochemical, rubber and shoe industries as well as in association with radiation exposure. Thus, the key to the allowance of claims is the existence of guidelines but the number of guidelines formulated lags well behind the accumulated knowledge of proven occupational carcinogens.

5. Furthermore, once the decision to accept a cause-and-effect relationship has been made, guidelines are developed based on the allowance of those circumstances (length and intensity of exposure, latency, etc.) which have been shown by particular epidemiological studies to be at a definitely increased risk. These numbers, however, are often arbitrarily chosen in the methodological design of the specific study. In some situations, (the cessation interval criteria, for example) the restrictions are actually based on very weak data and therefore are particularly unjustified. In other situations (the asbestos-related cancer guidelines, for example) the restrictions are even at odds with the recommendations of the scientists commissioned by the Board to study the situation. Often even "average" figures are used with seemingly little regard to standard deviations. Reviewing the scientific background to the 5 sets of guidelines which were detailed in this chapter suggests that often the requirements are needlessly demanding. The fact that the guidelines are occasionally revised in light of new scientific evidence, and cases are consequently re-adjudicated based on these updates, itself attests both to the importance of the guidelines and their overly restrictive nature. The time frame which characterizes this process is considerably lengthy. Furthermore, because this exercise of judgement takes place away from public scrutiny, the resulting guidelines are suspect. Consequently the very existence of numerical guidelines, as well as their particular criteria are understandably contentious.

Thus the problems relating to the guidelines are that there are too few of them relative to the existing knowledge; that the process of developing guidelines takes far too long; that the procedure is too secretive and lacks widespread input; and that those guidelines that do exist tend to be more restrictive than the state of scientific knowledge seems to support.

Possible reasons for this situation are discussed in Chapter V along with recommendations to address these difficulties.

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APPENDIX 1

B.C. Schedule of Diseases Re: Cancer

Cancer:

(a) Carcinoma of the lung when associated with asbestosis -
Where there is exposure to airborne asbestos dust.

(b) Mesothelioma (pleural or peritoneal) -
Where there is exposure to airborne asbestos dust.

(c) Carcinoma of the larynx or pharynx associated with asbestosis -
Where there is exposure to airborne asbestos dust.

(d) Gastro-intestinal cancer (including all primary cancers associated with the oesophagus, stomach, small bowel, colon and rectum (excluding the anus), and without regard to the site of the cancer in the gastro-intestinal tract or the histological structure of the cancer)

Where there is exposure to asbestos dust if during the period between the first exposure to asbestos dust and the diagnosis of gastro-intestinal cancer there has been a period of, or periods adding up to, 20 years of continuous exposure to asbestos dust and such exposure represents or is a manifestation of the major component of the occupational activity in which it occurred.

(e) Primary cancer of the lung

Where there is prolonged exposure to:
(1) aerosols and gases containing arsenic, chromium, nickel or their compounds; or
(2) bis (chloromethyl) ether; or
(3) the dust of uranium, or radon gas and its decay products; or
(4) particulate polycyclic aromatic hydrocarbons.

(f) Leukemia or pre-leukemia
Where there is exposure to benzene or to ionizing radiation.

(g) Primary cancer of the skin
Where there is prolonged contact with coal tar products, arsenic or cutting oils or prolonged exposure to solar ultra-violet light.

(h) Primary cancer of the epithelial lining of the urinary bladder, ureter or renal pelvis
Where there is prolonged exposure to beta-naphthylamine, benzidine, or 4-nitrodiphenyl.

(i) Primary cancer of the mucous lining of the nose or nasal sinuses
Where there is prolonged exposure to dusts, fumes or mists containing nickel or the dusts of hard woods.

(j) Angiosarcoma of the liver
Where there is exposure to vinyl chloride monomer.

APPENDIX 2
Uranium - Lung Cancer Claims Accepted (to end of 1979)

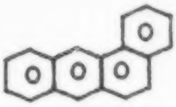
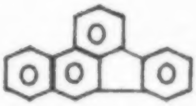
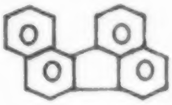
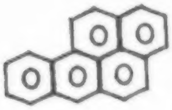
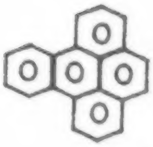
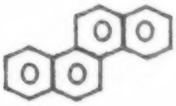
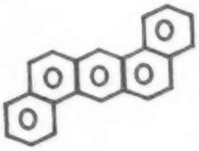
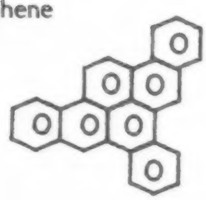
No.	Date Accepted	Age	Date Exposure	Death	Cancer Type	Latency	WLM
1.	1970	40	1957-68	Alive	Oat Cell	11 yrs.	110+
2.	1972	43	1958-71	1971	Oat Cell	13 yrs.	167+
3.	1972	40	1957-64	1972	Oat Cell	14 yrs.	175
4.	1973	48	1961-68 (42 mos.)	1970	Oat Cell	10 yrs.	111
5.	1974	54	1957-74	1974	Oat Cell	17 yrs.	134+
6.	1976	55	1955-73 (137 mos.)	1974	Oat Cell	18 yrs.	200
7.	1974	61	1957-73	1974	No Biopsy	17 yrs.	266
8.	1974	43	1957-73	1975	Squamous	16 yrs.	283
9.	1974	60	1957-69	1974	Squamous	16 yrs.	284
10.	1974	60	1958-64	1974	Squamous	16 yrs.	218
11.	1974	51	1957-63 (66 mos.)	1974?	Oat Cell	17 yrs.	190
12.	1974	58	1957-64 (84 mos)	1974	Sputum only	17 yrs.	70-90
13.	1975	59	1955-74	1974	N/A	18 yrs.	138+
14.	1975	37	1958-61 (45 mos.)	1961	Oat Cell	5 yrs.	130+
15.	1975	59	1954-60 1960-67	1974	N/A	19 yrs.	144
16.	1975	54	1957-64	1975	Anaplastic	17 yrs.	130
17.	1975	N/A	1957-62 (53 mos.)	1975?	N/A	17 yrs.	118
18.	1975	44	1956-68 (146 mos.)	Alive	Adeno Undiff.	18 yrs.	83+
19.	1975	52	1958-66	1970	Oat Cell	9 yrs.	230
20.	1975	57	1957-68	1970	Anaplastic	11 yrs.	209
21.	1976	53	1958-61 (30 mos.)	1974	Adeno	16 yrs.	90-104
22.	1976	47	1958-75 (190 Mos.)	N/A	Occult.	18 yrs.	87
23.	1976	59	1958-64	1975	Anaplastic	16 yrs.	
24.	1976	52	1958-64 1964-76	1976	Anaplastic	17 yrs.	157
25.	1976	52	1958-64 (60 mos.)	1975	Oat Cell	17 yrs.	46
26.	1976	55	1957-76 (213 mos.)	N/A	Poss. Oat.	18 yrs.	70+
27.	1977	53	1957-67 (106 mos.)	1976	Anaplastic	19 yrs.	235
28.	1976	53	1957-70 (156 mos.)	1970	Squamous	N/A	96.2
29.	1977	53	1955-67	1966	No Histology	10 yrs.	368
30.	1977	57	1957-63 (65 mos.)	1969	Oat Cell	11 yrs.	179.5
31.	1977	53	1957-63 (66 mos.)	1974	Oat Cell	N/A	159.76

No.	Date Accepted	Age	Date Exposure	Death	Cancer Type	Latency	WLM
32.	1977	61	1955-60 (52 mos.)	1971	Squamous	15 yrs.	90
33.	1977	60	1957-65 (78 mos.)	1976	Adeno Broncho.	18 yrs.	165.5
34.	1977	57	1958-75 (17 yrs.)	N/A	Oat Cell	17 yrs.	N/A
35.	1977	46	1957-65 (95 mos.)	1967	Oat Cell	9-10 yrs.	220
36.	1977	55	1955-57 (26 mos.)	1973	Squamous	17 yrs.	139
37.	1977	51	1957-75 (224 mos.)	N/A	Squamous	17 yrs.	102.36
38.	1977	59	1955-60 (57 mos.)	1974	Oat Cell	N/A	78.92
39.	1977	46	1954-61 (84 mos.)	1963?	N/A	N/A	375
40.	1977	43	1959-65 (6 years)	N/A	Adeno	N/A	139.20
41.	1976	63	1955-60 (64 mos.)	N/A	Adeno	22 yrs.	147.90
42.	1978	40	1956-62 (65 mos.)	1967	N/A	12 yrs.	143.27
43.	1977	61	1956-62 (67 mos.)	1972	Oat Cell	16 yrs.	N/A
44.	1978	62	1955-63 (90 mos.) 1967-77 (104 mos.)	N/A	Anaplastic	22 yrs.	130.41
45.	1978	42	1956-64 (62 mos.)	1972	Small Cell	16 yrs.	120.60
46.	1978	57	1957-62 (62 mos.)	1974	Squamous	14 yrs.	177.24
47.	1978	55	1958-69 (115 mos.)	N/A	Squamous	20 yrs.	72.5
48.	1978	70	1956-73 (196 mos.)	1978	Oat (Carcinoma)	21 yrs.	81.87
49.	1979	57	1958-70	N/A	Squamous	20 yrs.	113.47
50.	1979	50	1958-64 (79 mos.)	N/A	Oat Cell	20 yrs.	179
51.	1979	73	1958-64 (73 mos.)	N/A	N/A	N/A	117
52.	1979	44	1959-63	N/A	Squamous	17 yrs.	88
53.	1979	66	1957-61	N/A	Squamous	20+ yrs.	N/A
54.	1979	58	1955-65	N/A	Squamous	23 yrs.	68

APPENDIX 3

DATA ABOUT POLYCYCLIC AROMATIC HYDROCARBONS (INCLUDING COKE OVEN AND FOUNDRY STUDIES)

A. Indentified Polycyclic Aromatic Hydrocarbons

	Name	Chemical Structure	Animal Data	Human Data
1.	Benz(a)anthracene		- carcinogenicity by several routes of administration - lung, liver, skin & bladder cancer in mice - not adequately tested by other routes or in other species	- no studies (except in coal tar & other materials)
2.	Benzo(b)fluoranthene		- skin cancer in mice - not tested by other routes or in other species	as above
3.	Benzo(j)fluoranthene		as above	as above
4.	Benzo(a)pyrene		- both local & systemic cancer in all species tested (mouse, rat, hamster, guinea pig, rabbit, newt, monkey, & duck) following different administrations including oral, skin & intratracheal route.	as above
5.	Benzo(e)pyrene		- only tested by skin painting in mice: weaker than above - not tested by other routes or in other species	as above
6.	Chrysene		- as above: weakly carcinogenic	as above
7.	Dibenz(a,h)anthracene		- both local & systemic tumours incl. G.I. & lung by different routes of administration in all species tested (mice, rats, guinea pig, frogs, pigeons and chickens) - effective at lower dose than B(a)P, but longer latency period.	as above
8.	Dibenzo(h,rst)pentaphene		- in the only experiment reported caused cancer in mice	- only 1 study reports its occurrence in urban air

APPENDIX 3

DATA ABOUT POLYCYCLIC AROMATIC HYDROCARBONS (INCLUDING COKE OVEN AND FOUNDRY STUDIES)

B. Selected Tables from the Lloyd, Redmond *et al.* Steelworker Mortality Studies

Table A. Observed and Expected Mortality 1953-1966 and Relative Risk for workers in the Coke Plant in 1953 by Cause of Death and Place of Employment.

B. As above but including only those employed in the Coke Plant for 5 or more years.

C. Observed and Expected Mortality 1953-1966 and Relative Risk for Malignant Neoplasms by site, for Steelworkers employed at the Coke Ovens for 5 or more years.

D. As above, for workers in Non-Oven Areas of the coke plant.

E. Observed and Expected Mortality 1953-1975 and Relative Risk of Malignant Neoplasms for workers in the Coke Plant in 1953 by Cause of Death and Place of Employment.

F. As above including only those employed in the Coke Ovens.

G. By Cancer Sites for Workers Employed Five or More Years in Specified Work Areas for Allegheny County Steelworkers, 1953-1966+.

Table A

Observed and Expected Mortality, 1953-1966, and Relative [1] Risks for Allegheny County Steelworkers Employed in the Coke Plant in 1953 by Cause of Death and Place of Employment.

Cause of Death	COKE PLANT N=2543 Obs.	Exp.	Rel. Risk[1]	COKE OVEN N=1316 Obs.	Exp.	Rel. Risk[1]	NON-OVEN N=1227 Obs.	Exp.	Rel. Risk[1]
All Causes	397	367.1	1.10	199	170.1	1.14	198	185.2	1.08
Cancer of Respiratory System	45	25.9	1.93 **	34	12.0	3.19 **	11	11.6	0.95
Cancer of Lungs, Bronchus, Trachea	44	24.5	2.01 **	33	11.3	3.31 **	11	10.9	1.01
Cancer of Digestive Organs & Peritoneum	27	25.4	1.07	10	11.7	0.84	17	13.5	1.26
Cancer of Genitro-Urinary System	16	9.3	1.82 *	8	4.6	1.79	8	4.4	1.86
Prostate Cancer	8	5.6	1.48	4	2.9	--	4	2.6	--
Kidney Cancer	6	1.4	5.00 **	4	0.6	—	2	0.7	—
Bladder Cancer	2	2.0	--	0	1.0	--	2	0.9	--
Cancer of Lymph & Hematopoietic Tissues	6	6.2	0.96	2	2.8	—	4	3.1	—
Cancer of Buccal & Pharyngeal Organs	5	2.3	2.30	0	0.9	--	5	1.3	3.87 **
Cancer of Skin	0	0.6	—	0	0.2	—	0	0.3	—
All Other Cancers	9	8.5	1.07	5	4.2	1.20	4	4.0	--
Vascular Lesions of CNS	35	31.8	1.11	18	16.2	1.12	17	15.7	1.09
Arteriosclerotic Heart Disease	115	121.5	0.94	47	50.8	0.92	68	70.8	0.96
Other Heart Diseases	20	19.7	1.02	8	10.8	0.73	12	8.7	1.40
Other Respiratory Diseases	22	15.6	1.46	9	8.2	1.10	13	7.1	1.86 *
Accidents	25	23.8	1.06	15	13.2	1.14	10	10.6	0.95
All Other Causes	72	76.6	0.93	43	42.5	1.01	29	34.1	0.85

---

1 Relative risks compared to all other steelworkers.
* Significant at 5% level.
** Significant at 1% level.
-- Less than 5 deaths.

Table B

Observed and Expected Mortality, 1953-1966, and Relative Risks[1] for Allegheny County Steelworkers Employed in the Coke Plant for Five or More Years Through 1953 by Cause of Death and Place of Employment.

	TOTAL COKE PLANT N=1852			COKE OVEN N=965			NON-OVEN N=783			NO ONE COKE PLANT AREA N=104		
Cause of Death	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]
All Causes	402	366.5	1.12 *	214	192.4	1.14	178	163.5	1.11	10	9.6	1.05
Cancer of Respiratory System	48	26.5	2.05 **	41	14.8	3.53 **	6	10.3	0.58	1	0.6	—
Cancer of Lungs, Bronchus, Trachea	46	25.0	2.09 **	40	14.1	3.67 **	5	9.7	0.51	1	0.6	—
Cancer of Digestive Organs & Peritoneum	34	27.2	1.28	14	13.9	1.01	20	12.6	1.62 *	0	0.5	—
Cancer of Genito-Urinary System	17	10.3	1.76 *	10	5.8	1.81	7	4.1	1.73	0	0.2	—
Prostate Cancer	8	6.3	1.31	5	3.6	1.42	3	2.5	—	0	0.1	—
Kidney Cancer	6	1.6	4.50 **	4	0.8	—	2	0.7	—	0	0.0	—
Bladder Cancer	3	2.2	—	1	1.3	—	2	0.9	—	0	0.0	—
Cancer of Lymph & Hematopoietic Tissues	7	6.4	1.11	3	3.3	—	4	2.8	—	0	0.2	—
Cancer of Buccal & Pharyngeal Organs	5	2.5	2.14	1	1.0	—	4	1.3	—	0	0.1	—
Cancer of Skin	0	0.5	—	0	0.2	—	0	0.2	—	0	0.0	—
All Other Cancers	8	8.6	0.93	6	4.9	1.26	2	3.5	—	0	0.3	—
Vascular Lesions of CNS	41	34.0	1.23	20	18.6	1.08	20	14.6	1.39	1	0.7	—
Arteriosclerotic Heart Disease	118	126.5	0.93	48	58.8	0.80	64	64.4	0.99	6	3.3	1.89
Other Heart Diseases	21	20.5	1.03	8	11.7	0.66	13	8.0	1.66	0	0.6	—
Other Respiratory Diseases	21	16.1	1.34	11	9.3	1.20	10	6.4	1.60	0	0.4	—
Accidents	20	16.8	1.21	11	9.4	1.19	8	6.6	1.22	1	0.7	—
All Other Causes	62	70.8	0.86	41	40.5	1.01	20	28.7	0.69	1	2.1	—

1. Relative risks compared to all other steelworkers.
* Significant at 5% level.
** Significant at 1% level.
— Less than 5 deaths.

Table C

Observed and Expected Mortality, 1953-1966, and Relative Risks[1] for Malignant Neoplasms by Site for Allegheny County Steelworkers Employed at the Coke Ovens for Five or More Years Through 1953.

	COKE OVEN EXPERIENCE			OVEN TOPSIDE FULLTIME			OVEN TOPSIDE PART-TIME			OVEN SIDE		
	N=965			N=149			N=177			N=639		
Cause of Death	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]
Malignant Neoplasms - All Sites	75	43.9	1.85 **	28	7.8	4.03 **	11	7.1	1.56	36	26.8	1.38
Respiratory Organs	41	14.8	3.53 **	20	2.6	10.83 **	3	2.3	—	18	8.1	2.[illegible]
Digestive Organs & Peritoneum	14	13.9	1.01	1	2.4	—	4	2.5	—	9	9.0	1.00
Stomach	6	5.1	1.18	1	1.0	—	2	0.7	—	3	3.4	—
Large Intestine	3	3.4	—	0	0.6	—	1	0.6	—	2	2.3	—
Rectum	1	1.2	—	0	0.2	—	0	0.3	—	1	0.7	—
Pancreas	2	1.7	—	0	0.3	—	0	0.3	—	2	1.1	—
Other Digestive Organs	2	2.4	—	0	0.4	—	1	0.6	—	1	1.5	—
Genito-Urinary Organs	10	5.8	1.81	1	1.1	—	2	0.7	—	7	4.0	1.86
Prostate	5	3.6	1.42	1	0.7	—	1	0.4	—	3	2.5	—
Kidney	4	0.8	—	0	0.1	—	1	0.1	—	3	0.5	—
Bladder and Other Organs	1	1.4	—	0	0.3	—	0	0.2	—	1	0.9	—
Lymph & Hematopoietic Tissues	3	3.3	—	1	0.5	—	1	0.6	—	1	2.0	—
Buccal & Pharyngeal Organs	1	1.0	—	0	0.1	—	1	0.2	—	0	0.7	—
All Other Cancers	6	5.1	1.19	5	1.0	6.09 **	0	0.8	—	1	3.0	—

1. Relative risks compared to all other steelworkers.
* Significant at 5% level.
** Significant at 1% level.
— Less than 5 deaths.

Table D

Observed and Expected Mortality, 1953-1966, and Relative Risks[1] for Malignant Neoplasms by Site for Allegheny County Steelworkers Employed in Non-Oven Areas of the Coke Plant for Five or More Years Through 1953.

	NON-OVEN EXPERIENCE N=783			BY-PRODUCTS N=345			COAL HANDLING N=111			ALL OTHER COKE PLANT NON-OVEN N=327		
Cause of Death	**Obs.**	**Exp.**	**Rel. Risk[1]**	**Obs.**	**Exp.**	**Rel. Risk[1]**	**Obs.**	**Exp.**	**Rel. Risk[1]**	**Obs.**	**Exp.**	**Rel. Risk[1]**
Malignant Neoplasms - All Sites	43	34.8	1.25	10	13.5	0.73	11	6.1	1.83	22	15.0	1.48
Respiratory Organs	6	10.3	0.58	1	4.0	--	2	1.9	--	3	4.5	--
Digestive Organs & Peritoneum	20	12.6	1.62 *	4	4.8	--	6	2.2	2.74 *	10	5.4	1.88
Stomach	3	3.6	--	0	1.4	--	2	0.6	--	1	1.5	--
Large Intestine	9	3.2	2.93 **	2	1.2	--	3	0.6	--	4	1.4	--
Rectum	0	1.4	--	0	0.5	--	0	0.3	--	0	0.6	--
Pancreas	7	1.7	4.55 **	2	0.6	--	1	0.3	--	4	0.7	--
Other Digestive Organs	1	2.7	--	0	1.0	--	0	0.5	--	1	1.2	--
Genito-Urinary Organs	7	4.1	1.73	2	1.7	--	2	0.7	--	3	1.7	--
Prostate	3	2.5	--	1	1.0	--	1	0.4	--	1	1.0	--
Kidney	2	0.7	--	0	0.3	--	1	0.1	--	1	0.3	--
Bladder and Other Organs	2	1.0	--	1	0.4	--	0	0.2	--	1	0.4	--
Lymph & Hematopoietic Tissues	4	2.8	--	2	1.1	--	0	0.5	--	2	1.2	--
Buccal & Pharyngeal Organs	4	1.3	--	0	0.5	--	1	0.2	--	3	0.6	--
All Other Cancers	2	3.7	--	1	1.5	--	0	0.6	--	1	1.6	--

1. Relative risks compared to all other steelworkers.

* Significant at 5% level.

** Significant at 1% level.

-- Less than 5 deaths.

Table E

Observed and Expected Mortality, 1953-1975, and Relative Risks[1] for Allegheny County Steelworkers Ever Employed in the Coke Plant in 1953 by Cause of Death and Place of Employment.

	COKE PLANT N=3558			COKE OVEN N=2041			NON-OVEN N=1517		
Cause of Death	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]
All Sites-Malignant Neoplasms	294	249.2	1.21 **	179	144.4	1.29 **	115	102.8	1.13
Respiratory Organs	121	83.4	1.55 **	90	50.1	2.03 **	31	32.4	0.95
Lungs, Trachea, Bronchus	114	78.7	1.55 **	86	47.4	2.05 **	28	30.6	0.91
Other Respiratory Organs	7	4.6	1.63	4	2.6	—	3	1.8	—
Digestive Organs and Peritoneum	84	79.6	1.06	34	44.4	0.74	50	34.6	1.48 **
Large Intestine	24	21.1	1.15	6	11.1	0.51	18	9.8	1.90 *
Pancreas	18	12.3	1.54	5	6.8	0.71	13	5.1	2.71 **
Other Digestive Organs	42	46.2	0.90	23	26.5	0.86	19	19.7	0.96
Genito-Urinary Organs	41	32.2	1.32	28	18.9	1.56 *	13	12.8	1.02
Prostate	29	21.1	1.45	20	12.7	1.67 *	9	7.8	1.16
Kidney	8	4.5	1.89	7	2.6	2.88 *	1	1.8	—
Bladder	3	5.8	0.50	1	3.2	—	2	2.7	—
Buccal Cavity and Pharyngeal Organs	8	7.4	1.09	2	3.9	—	6	3.4	1.81
Lymph and Hematopoietic Tissues	18	18.6	0.97	10	10.7	0.92	8	7.8	1.02
Skin Cancer	0	1.6	—	0	0.8	—	0	0.8	—
All Other Cancers	22	26.5	0.81	15	15.7	0.95	7	10.9	0.63

* Significance of Relative Risk (Rel. Risk) based on Summary Chi-Square with One Degree of Freedom.
* Significance at 5% level.
** Significance at 1% level.
— Less than 5 deaths.

Table F

Observed and Expected Mortality, 1953-1975, and Relative Risks[1] for Malignant Neoplasms By Site for Allegheny County Steelworkers Ever Employed at the Coke Ovens Through 1953.

	Coke-Oven Experience			Oven Topside Full-Time			Oven Topside Part-Time			Oven Side		
	N=2041			N=305			N=320			N=1416		
Cause of Death	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]	Obs.	Exp.	Rel. Risk[1]
All Sites-Malignant Neoplasms	179	144.4	1.29 **	56	25.9	2.37 **	25	21.9	1.15	98	92.9	1.06
Respiratory Organs	90	50.1	2.03 **	36	9.0	4.79 **	9	7.2	1.25	45	30.4	1.56 **
Lungs, Trachea, Bronchus	86	47.4	2.05 **	35	8.6	4.87 **	8	6.8	1.17	43	28.7	1.58 **
Other Respiratory Organs	4	2.6	--	1	0.4	--	1	0.4	--	2	1.7	--
Digestive Organs and Peritoneum	34	44.4	0.75	4	7.8	0.50	5	7.2	0.70	25	29.8	0.83
Large Intestine	6	11.1	0.51	0	1.9	--	1	2.0	--	5	7.4	0.65
Pancreas	5	6.8	0.71	1	1.2	--	0	1.0	--	4	4.6	--
Other Digestive Organs	23	26.5	0.86	3	4.6	--	4	4.2	--	16	17.8	0.89
Genito-Urinary Organs	28	18.9	1.56 *	6	3.7	1.70	6	2.5	2.50	16	12.4	1.32
Prostate	20	12.7	1.67 *	4	2.5	--	3	1.4	--	13	8.5	1.60
Kidney	7	2.6	2.88 *	2	0.5	--	3	0.4	--	2	1.6	--
Bladder	1	3.2	--	0	0.6	--	0	0.6	--	1	2.2	--
Buccal Cavity and Pharyngeal Organs	2	3.9	--	0	0.6	--	1	1.7	--	1	2.7	--
Lymph and Hematopoietic Tissues	10	10.7	0.92	3	1.8	--	1	0.7	--	6	7.0	0.84
Skin Cancer	0	0.8	--	0	0.1	--	0	0.2	--	0	0.6	--
All Other Cancers	15	15.6	0.95	7	3.0	2.50 *	3	2.4	--	5	10.0	0.49

1. Significance of Relative Risk (Rel. Risk) based on Summary Chi-Square with One Degree of Freedom.
* Significant at 5% level.
** Significant at 1% level.
-- Less than 5 deaths.

Table G

By Cancer Sites For Workers Employed Five or More Years in Specified Work Areas
Allegheny Steelworkers, 1953 - 1966+

Site	I.C.D. List Numbers	Work Area	Observed	Expected	Relative Risk
All Cancers	140-205	Coke Plant	119	91.9	1.51 **
Digestive Organs	150-159	Stainless Annealing	6	2.3	2.80 *
Esophagus	150	Open Hearth	13	6.6	2.41 *
Stomach	151	Blast Furnace	17	12.3	1.45
Large Intestine	153	Coke Plant	12	7.8	1.77
Rectum	154	Machine Shop	6	2.5	2.55
Respiratory System	160-164	Coke Plant	45	34.9	1.81 **
		Mason Department	17	11.0	1.57
		Blacksmith Shop	8	3.8	2.16
		Machine Shop	24	16.6	1.48
Genito-Urinary Organs	177-181	Coke Plant	17	10.4	2.01 *
		Janitors	7	3.4	2.28
Prostate	177	Janitors	6	2.3	3.11 *
Kidney	180	Coke Plant	6	1.5	15.20 **
Leukemia & Lymphoma	200-205	Heat Treating & Forging	5	2.2	2.87

Source: Carol K. Redmond, 1975. Comparative Cause-Specific Mortality Patterns by Work Area within the Steel Industry. U.S. Department of Health, Education and Welfare, Public Health Service, CDC, NIOSH, HEW Publication No. (NIOSH) 75-157.

* Significant at 5% level.
** Significant at 1% level.

C. Selected Tables from Gibson's Foundry Mortality Study

Table A

Standardized Mortality Ratios for Foundry and Non-Foundry Workers

	All Causes		All Cancer		Lung Cancer	
	Foundry	Non-Foundry	Foundry	Non-Foundry	Foundry	Non-Foundry
Observed	95.00	199.00	37.00	48.00	18.00	10.00
Expected	105.95	208.26	26.75	53.27	8.40	16.58
S.M.R.	90.00	96.00	138.00	90.00	214.00*	60.00
X^2	1.13	0.42	3.93*	0.52	10.97**	2.79

* Significant at 0.05; ** Significant at 0.005

Table B

Deaths by Foundry Job Class

	Cranes	Finishing	Coremaking	Moulding	Electric Furnace/ Open Hearth
a) All Deaths					
Expected	7.49	26.90	12.02	19.19	31.11
Observed	9.00	30.00	14.00	15.00	20.00
S.M.R.	121.00	115.00	116.00	78.00	64.00
b) All Cancer Deaths					
Expected	1.85	7.02	3.07	4.97	8.24
Observed	6.00	12.00	4.00	7.00	5.00
S.M.R.	324.00	171.00	130.00	141.00	61.00
c) Lung Cancer					
Expected	0.56	2.23	0.96	1.57	2.64
Observed	3.00	6.00	2.00	3.00	3.00
S.M.R.	536.00*	269.00	208.00	191.00	114.00

* Significant at .05

Table C

Dose Response - Years of Exposure and Lung Cancer Deaths

Exposure	Number Employees	Person Years at Risk	Expected Lung Cancer Deaths	Observed	X^2
1-59 months	188	1759.09	2.45	1	0.86 (N.S.)
5 Years or More	439	3929.98	8.40	18	10.97*

* Significant at .005

Table D
Lung Cancer Deaths
Dose Response - Twenty Years Since Initial Exposure, Compared to All Other Foundry Employees

	Number	Person Years at Risk	Expected Deaths	Observed Deaths	S.M.R.	X^2 (1)
Twenty Years Since First Exposure	341	2998.76	7.53	17	226*	11.91
All Others	98	931.63	0.88	1	113	

* Significant at 0.05

Table E - Claims Analysis in the Hamilton Foundry

Claims	Exposure	Latency	Cessation Interval
1	16 yr. 5 mo. (16.42)	17	0
2	20 yr. 7 mo. (20.58)	38	17
3	9 yr. 9 mo. (9.75)	27	17
4	38 yr. 1 mo. (38.08)	53	14
5	3 yr. 9 mo. (3.75)	16	4
6	5 yr. 10 mo. (5.83)	10	4
7	11 yr. 3 mo. (11.25)	26	14
8	41 yr. 5 mo. (41.42)	32	0
9	14 yr. 10 mo. (14.83)	40	25
10	12 yr. 3 mo. (12.25)	30	18
11	21 yr. 9 mo. (21.75)	32	8
12	15 yr. 2 mo. (15.17)	35	20
13	7 yr. 4 mo. (7.33)	32	25
14	30 yr. 7 mo. (30.58)	31	0
15	24 yr. 5 mo. (24.42)	25	1
16	29 yr. 10 mo. (29.83)	34	4
17	26 yr. 4 mo. (26.33)	33	6
18	23 yr. 5 mo. (23.42)	35	12
19	34 yr. 6 mo. (34.50)	34	0
20	31 yr. 3 mo. (31.25)	39	8
21	22 yr. 4 mo. (22.33)	36	14
22	20 yr. 8 mo. (20.65)	38	17
23	26 yr. 9 mo. (26.75)	37	10
Low	3 yr. 9 mo.	10	0
Mean	21 yr. 3 mo. (21.23)	31.73	10.78
High	41 yr. 5 mo.	53	25

APPENDIX 4

Data from Mortality Studies of Nickel Refinery Workers in Ontario

Table A

Mortality Study of Nickel Refinery Workers, Copper Cliff
Sutherland, 1968

Reported vs Expected Deaths 1948 - June 1968[2] by Duration of Exposure

	Exposure 0-4+ Years[b]	5-10 Yrs	10+ Yrs	Durations Combined
Pulmonary Cancer				
Reported:	1	1	5 **	7 **
Expected:	0.23	0.23	0.32	0.78
Non-Pulmonary Cancer				
Reported:	-	-	1	1 [c]
Expected:	1.01	0.77	0.87	2.65

** 1% level of significance.

Notes

a. Follow-up period extended only 15 years after termination of employment (i.e. cancers with latent periods of longer than 15 years may have been missed.)

b. Cohort was defined as those with exposure of 5 years or more, therefore this group may be incomplete (i.e. cancers in those with less than 5 years of exposure may have been missed).

c. This was a case of proven cancer of the nasal sinus. (Had the expected rate for nasal sinus rather than all non-pulmonary cancer been used, the SMR would have been much higher.)

Table B

Review of Mortality of Nickel Refinery Workers, Copper Cliff, Chovil, 1980

Relationship of Period of First Employment to the Development of Primary Lung Cancer

Year of First Employment	1948-51	1952-62
No. of Workers	279	216
Observed Deaths (Ca. lung)[a]	26	1
Expected Deaths (Ca. lung)	3.17	1.08
SMR	11.36	0.93
Observed Cases (Ca. lung)	51	3
Expected Cases (Ca. lung)	4.76	1.62
SIR	10.71	1.85

Notes

a Table Includes All Cases Reported Up to December 1978

b SIR "Standard Incidence Ratio" using a factor of 1.5 to adjust expected deaths to expected cases.

Table C
Tabulation of Selected Results from Roberts et al. (JOHC), 1982: Nasal and Lung Cancer in Sinter Plant Workers

	All Sinter Plant Workers in Cohort			Sinter Plant Workers With More Than 15 Years Since First Exposure					
				5 Years Exposure			5 Years Exposure		
	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
A. Nasal Cancer									
Sudbury Sinter	2	0.4	1,463 ***	1	0.07	1,429	1	0.02	4,545
P.C. (L.C. & S.)[a]	16	0.20	8,000 ***	3	0.09	3,297 ***	13	0.08	16,883 ***
B. Lung Cancer									
Sudbury Sinter	46	11.34	406 ***	18	6.52	276 ***	24	2.57	935 ***
Copper Cliff	41	9.68	424 ***	17	5.84	291 ***	20	1.88	1,067 ***
Coniston	5	1.75	286 *	1	0.72	140	4	0.69	581 ***
P.C. (L.C. & S.)[a]	50	17.90	279 ***	17	9.24	184 ***	32	7.20	445 ***

*	P / 0.05
**	P / 0.01
***	P / 0.001

Notes

a - L, C & S: Leaching, calcining and sintering

APPENDIX 5

GUIDELINES FOR THE ALLOWANCE OF ASBESTOS - RELATED CANCERS AND STUDIES OF OCCUPATIONAL LARYNGEAL CARCINOMA

A. WCBO Guidelines for the Allowance of Asbestos-Related Cancers.

1. Claims for mesothelioma are favourably considered when:

    1. There is a clear and adequate history of at least 10 years occupational exposure to asbestos; and

    2. There is a minimum interval of 15 years between first exposure to asbestos and the appearance of mesothelioma.

    3. Claims which do not meet the guidelines in (1) and (2) should be individually judged on their own merit...

2. Claims for lung cancer are favourably considered when:

    1. There is a clear and adequate history of at least 10 years occupational exposure to asbestos; and

    2. There is a minimum interval of 10 years between first exposure to asbestos and the appearance of lung cancer.

    3. Claims which do not meet the guidelines in (1) and (2) should be individually judged on their own merit...

3. Claims for gastro-intestinal cancer are favourably considered when:

    1. There is a clear and adequate history of exposure to asbestos "of a continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity."

    2. There is a minimum interval of 20 years since first exposure.

    3. Claims which do not meet the guidelines in (1) and (2) should be individually judged on their own merit...

4. Claims for cancer of the larynx: See Page 247.

B. Summary of Reports on Occupational Laryngeal Carcinoma Available at the Time the Guidelines were Formulated

Author(s)	Year	Study Design	Numbers Involved	Findings
Stell and McGill	1973 (Lancet 2:416)	case-control	100 pairs (cancer of the head and neck and controls)	- highly significant difference between throat cancer rates in asbestos-exposed and non-exposed groups (31% vs 3%)
Stell and McGill	1973 (Lancet 2:678)	case-control	100 pairs (cancer of larynx and controls)	- relative risk of 14.5 for asbestos-related throat cancer - (smoking a co-factor?)
Newhouse and Berry	1973 (Lancet 2:615)	cohort-mortality	4,000	- two deaths from laryngeal cancer when 0.37 expected, among asbestos-exposed cohort members - assumed both smokers (R.R. = 5.4)
Pederson et al.	1973 (Int. J. Cancer 12:32)	follow-up	1916 men	- 5 laryngeal cancers found compared to 1.29 expected highly significant (S.M.R. 13.6); exposure: nickel refinery (roasting and smelting) - (no smoking history)
Libshitz et al.	1974 (JAMA 228: 1571)	cases	3 men	- all had asbestosis, epidermoid laryngeal cancer and a smoking history - possible relationship between asbestos exposure and throat cancer (smoking a co-factor?)

Author(s)	Year	Study Design	Numbers Invloved	Findings
McDonald et al.	1974 (not published until 1980, Br. J. Ind. Med. 37:11)			- early results showed no excess risk to chrysotile asbestos miners - R.R. = 1.07
Stell and McGill	1975 (Laryngol, Rhinol. Otol. 89: 513)	hospital case-control	119 (cancer of larynx, patient and controls)	- all squamous cell throat cancers - (highly significant difference) - 27.7% of cancer cases had asbestos exposure versus 2.5% of controls, $X^2 = 25$, p< .001) - asbestos link to throat cancer established, at least excess risk - (smoking a co-factor?)
Shettigara and Morgan	1975 (Arch. Environ. Hlth. 30:517)	neighbourhood controls and hospital cases	43 pairs	- substantial association between asbestos exposure and laryngeal cancer (23.3% of cases had asbestos exposure vs 0 controls) - (smoking a co-factor?) - no correlation of excess risk with exposure to nickel, uranium, chromium, cobalt, arsenic, x-rays or alcohol
Selikoff et al.	1975 (Table first appeared in 1975; in "Persons at High Risk of Cancer," Fraumeni ed., Acad. Press, N.Y.; presented in Symposium 1976, published separately 1977)	cohort mortality	17,800	- observed 9 deaths from laryngeal cancer when 3.81 were expected (SMR 2.36) Bucco-pharyneal cancer 1.91 times expected - (smoking a co-factor?)

Author(s)	Year	Study Design	Numbers Involved	Findings
Morgan and Shettigara	1976 (Ann. N.Y. Acad. Sci 271: 308)	neighbourhood controls and hospital cases	54 pairs, including previous 43	- 24% of laryngeal cancer cases and almost no controls had asbestos exposure; (R.R. = 13) - confirms relation of asbestos to laryngeal cancer, for smokers
Wynder et al.	1976 (Cancer 38: 1591)	case-control	258 men and 56 women	- excess smoking and alcohol effect - excess occupational exposure (asbestos) not quantified
Freifeld	1977 (JAMA 238: 1280)	case	one	- epidermoid cancer of vocal cord in non-smoker with 30 years asbestos exposure
Hinds	1979 (Cancer 44:1114)	case-control	47 pairs	- asbestos-throat cancer relative risk of 1.75, "not significant" - (questions importance of asbestos exposure in causing this cancer) - no relationship either between throat cancer and number of other occupational exposures - smoking and alcohol independently increase risk of throat cancer, with dose-response relationship - so too, do dental x-rays among heavy smokers
Hirsch et al.	1979 (Chest 76: 6)	cases	two	- found asbestos fibres in patients, one with throat cancer, other with polyp in throat

Author(s)	Year	Study Design	Numbers Involved	Findings
Newhouse et al.	1980 (IARC 2:30)	descriptive study (pathological specimens)	305 men, 206 women	- insignificant exposure difference between those with neoplasia and other groups - neoplasia associated with cigarette smoking alone but alcohol independent of smoking showed no increased effect
Blot et al.	1980 (JNCI 65:571)	case-control deaths	64 laryngeal cancer (361 controls)	- tobacco and alcohol were major risk factors among east coast American shipyard workers - results for asbestos were mixed, with relative risk of getting throat cancer 2.4 for blacks and slightly less than 1 for whites - blacks had half the laryngeal cancers but only 1/3 of the lung cancers
Blair and Mason	1980 (Arch. Environ. Hlth. 35: 92)	national cancer mortality ratios	rates per population in thousands	- electroplating industry workers have higher risk of larynx cancer (and other cancers) than in areas without the industry - must study to find cause(s)

C. Conclusions From Miller's Interim Report, 1978

"The following conclusions seemed justified from the analysis of our preliminary results and the previously reported data in the world literature.

1. Exposures of the type which may well be relevant to the etiology of laryngeal cancer are not identified through routine occupational histories. Special questioning is required in order to elicit information on such exposure. There is, however, no evidence that can be identified of recall bias in our method of questioning.

2. There has been a greater exposure to asbestos encountered occupationally in cases than controls with a risk ratio of the order of 2 to 4. This is not due to confounding by other factors with the exception of joint exposure to asbestos and nickel in three cases. The numbers available in the present analysis do not suffice to evaluate the association of asbestos and laryngeal cancer on its own. Nevertheless, the risk ratio is of a similar order to that identified by Selikoff in his cohort studies though substantially less than that reported by Morgan and Shettigara.

3. There is a signficantly greater exposure to nickel in cases than controls. This is not due to confounding except for the joint exposure between asbestos and nickel exposure and is compatible with the only other previously reported study, that of Pedersen and his colleagues.

4. The elucidation of other relevant etiological factors with regard to both asbestos and nickel exposure and cancer of the larynx requires further study and, in particular, a larger series of cases and controls. Thus, the data do not permit unravelling of the joint exposure to both agents nor is it possible at this stage from the data to determine the severity of exposure which is required to be etiologically relevant nor the appropriate latent period. On the other hand, the data currently available suggests that some of the previously identified relationships of asbestos and other cancers with, for example, a 15 to 20 year latent period and an established period of exposure over several months if not years is likely to be important."

D. Conclusions from Miller's Final Report, 1981

1. "Exposure to asbestos is associated with the subsequent development of cancer of the larynx. The association is not due to confounding from cigarette exposure or alcohol consumption. Asbestos exposure must therefore be regarded as a cause of cancer of the larynx.

2. The latent period from first exposure to development of cancer of the larynx should be 10 years or more for the exposure to be regarded as causal.

3. The degree of increased risk of cancer of the larynx following occupational exposure to asbestos approximates to two-fold.

4. There is currently no convincing evidence that nickel exposure is a cause of cancer of the larynx. However this has been suggested in one cohort study of occupationally exposed workers in Norway, and further consideration of this issue when new data are available on Canadian workers will be necessary."

CHAPTER III

NON-MALIGNANT OCCUPATIONAL RESPIRATORY DISEASE

A. ISSUES AND DIMENSIONS OF CHRONIC OCCUPATIONAL RESPIRATORY DISEASE

1. Introduction

1.1 Overview

Respiratory disease is a serious problem in Ontario as in many temperate regions. As a major cause of illness and suffering it also accounts for a significant number of work absences and hospital admissions. Moreover, the number of people dying from chronic respiratory disease is on the increase in Canada.

It is not easy to determine the extent to which workplace conditions contribute to these disabilities. Some occupational lung diseases are well-known and readily recognizable as job-related, such as asbestosis and silicosis. However, many victims of occupational lung disease do not have a clearly defined occupation-specific disease. Instead they have bronchitis, asthma, emphysema, or cancer - conditions that can be caused or aggravated by many different occupations as well as non-occupational factors such as smoking and pollution. Exposure information needed to evaluate the extent of workplace respiratory hazards as well as to assess non-occupational factors is almost always incomplete. Apportioning cause in the individual case, has therefore, little validity and may be subject to judgemental bias or to arbitrary decision making.

Furthermore, different diseases often have similar symptoms and diseases overlap making precise diagnosis difficult. "Objective tests" have their limitations; impairment and disability are not synonymous, hence "disability assessments" are also subject to controversy. Many policy decisions have to be made regarding what is or is not compensable. Thus respiratory disease is a difficult area for workers' compensation systems. The vast numbers of people afflicted and the serious effect these diseases can have on their victims makes them an important area meriting careful scrutiny.

Part A of this chapter will discuss the issues involved in assessing respiratory disease. Some historical background, will be presented first. Section 2 will discuss the potential scope of the problem by outlining the prevalence of respiratory disease in Ontario. Some basic information will be provided about the respiratory system in health and disease to assist the non-medical reader in appreciating the complexity of the compensation dilemma. Issues pertinent to compensation will be identified and the range of pathological reactions will be introduced. Section 3 will review the general state of knowledge on the nature and prevalence of work-related respiratory diseases and Section 4 will discuss some of the difficulties encountered in diagnosis and assessment. Part B of this chapter will discuss how respiratory diseases are handled in the workers' compensation system in Ontario.

1.2 Historical Background

The problem of occupational lung disease is not new - in fact, it has been recognized for over four centuries. Moreover, the need for engineering controls in industry to prevent lung disease was stressed over four centuries ago. Although some occupational lung disease probably existed from the beginning of human history, the advent of mining produced the first recorded cases of occupational respiratory disease.

Silver and gold were first discovered around AD 1000 in the Hartz mountains of Silesia and Bohemia, and it was in these metal mines that the first observations were made that led to the recognition of dust as an industrial hazard.[1] In the mid-sixteenth century Paracelsus observed that cough, shortness of breath and wasting diseases were more prevalent in certain mines, but attributed these symptoms merely to "the workings of the gods."[2] In 1556 Agricola first related these effects to the dust and recommended the use of ventilating machines.[3] About a century later, Bernardino Ramazzini, the father of occupational medicine, brought further light to the health problems and work conditions of numerous occupations.[4]

With the advent of the industrial revolution, the prevalence of industrial disease increased significantly. Although no morbidity and mortality statistics were kept, the appalling conditions quickly became known.[5] In 1838, Louis found that 23 of 50 patients dying of cholera had some degree of what he called "emphysema"[6]. Dickens and others graphically described the horrors of mill and factory working life. In Sheffield, cutlery trade workers were dying of silicosis, known then as grinders rot, with as little as five years exposure.[7] The cries for industrial reform could not be stifled; workers demanded improved conditions and recourse for the ills inflicted upon them by their jobs.

After a period of unsatisfactory experience with tort litigation in the compensation of industrial injury, workers' compensation was adopted at the turn of the century. Although a tremendous social advance, problems remained. Coverage was not extended to all workers, disability awards varied considerably from place to place, and even many well-recognized industrial diseases were not always covered. Enforcement was also problematic, e.g. Morgan describes the death of 400 workmen from acute silicosis while working in the Gauly Bridge Tunnel in West Virginia between 1932 and 1934. The bodies were buried in secret so that exhumation was impossible and not a single worker or his or her family received compensation.[8]

Many problems in the compensation system persist to this day, and are being compounded by the ever increasing list of occupational hazards to the respiratory system. Occupational respiratory disease, one of the first recognized consequences of industrial exposure, is still very much a growing concern.

2. An Overview of Respiratory Diseases

2.1 Prevalence of Respiratory Disease

It is difficult to assess the prevalence of occcupational respiratory diseases with any degree of certainty. First, the issue of what constitutes an "occupational" lung disease is hotly debated; secondly the diagnostic criteria of each disease are not uniform; thirdly, the number of workers exposed to respiratory hazards, the duration and intensity of these exposures, dose-response data and relative risk relationships between exposure and disease are all sorely lacking; lastly, the reporting systems are embryonic at best. It is, in fact, well-known that workers' compensation statistics grossly underestimate the actual situation,[9] although there is much debate over how vast the problem really is.

Knowledge of the extent of respiratory diseases as a whole, however, is a useful starting point. Respiratory diseases account for a large number of hospital admissions. More than 143,000 hospitalizations in Ontario in 1976 were due to respiratory disease, making it one of the leading causes of hospital morbidity.[10] Moreover, about 3,000 people die annually of respiratory causes in Ontario (including merely those in whom the cause of death was listed as influenza, pneumonia, bronchitis, emphysema and asthma), making it one of the 5 leading causes of death in this province.[11] (See Appendix 1 of Chapter I, Part B.)

Trends in respiratory disease mortality are also remarkably revealing. Age-standardized mortality rates for non-neoplastic respiratory disease declined by 8% for males and 24% for females between 1969 and 1978, with most of the decrease being due to declining rates of pneumonia. However, mortality rates for bronchitis, emphysema and asthma increased by 21% for males and 38% for females[12] (see Appendix 1, Table A). Age-specific mortality rates for respiratory diseases declined in all age groups for women and up to age 75 for men; with older men experiencing slightly increasing rates (see Appendix 1, Table B). It is stated that the parallel increase in mortality rates for bronchitis, emphysema and asthma with lung cancer mortality rates suggests that those changes are attributable primarily to smoking since the latter is the strongest risk factor for both disease categories. However, due to inadequate study of occupational variables, also potentially common to both disease categories, attributing these trends solely to smoking may in fact prove to be overly simplistic. Although smoking is the most important variable, the occupational component should not be ignored. As shown in Chapter II, the evidence regarding the role of the workplace in malignant respiratory diseases has increasingly been proven correct. Perhaps, too, the role of occupational factors in non-malignant respiratory diseases will also see increasing recognition. In fact, just as the debate around the occupational contribution to lung cancer was the issue of the seventies, the role of occupational exposures in non-malignant respiratory disease may well be the major debate of the eighties.

One cannot draw causal inferences from the statistics, although they do serve to illustrate the enormous scope of the respiratory disease problem.

2.2 *Structure and Function of the Respiratory System*[13]

In order to appreciate the complexities in establishing an accurate diagnosis and assessing the extent of disability caused by the various occupational lung conditions, it is necessary to have some understanding of how these diseases produce respiratory impairment. It is therefore useful to discuss briefly the structure and function of the respiratory system in health and disease.

The main task of the lungs is to supply oxygen needed by the body and to remove carbon dioxide (a waste product) produced as body cells use oxygen. The lungs must keep the oxygen and carbon dioxide content of the arterial blood within a narrow range despite the constant fluctuation of oxygen requirements and carbon dioxide production with changes in degree of activity. This function of gas exchange occurs in the 300,000,000 or so microscopic air sacs called "alveoli" located deep within the lungs. Here, inhaled air is separated from the bloodstream by only a very thin delicate membrane and can normally pass readily into the blood.

The respiratory system is represented diagramatically in Figure IIIA-1. Air travels from the nose and mouth to the alveoli through a network of progressively branching tubes. The largest tube, the trachea, leads from the throat into the chest, where it divides into two main bronchi leading into each of the two lungs. Each bronchus branches into smaller bronchi, which then branch repeatedly into bronchioles, the smallest of which end at the alveoli. Gas exchange takes place in the last generations of bronchioles called "respiratory bronchioles", as well as in the alveoli themselves. The first twelve or so divisions all have stiff but resilient cartilage rings in their walls which help to prevent these airways from collapsing. Millions of bronchioles beyond these have no cartilage.

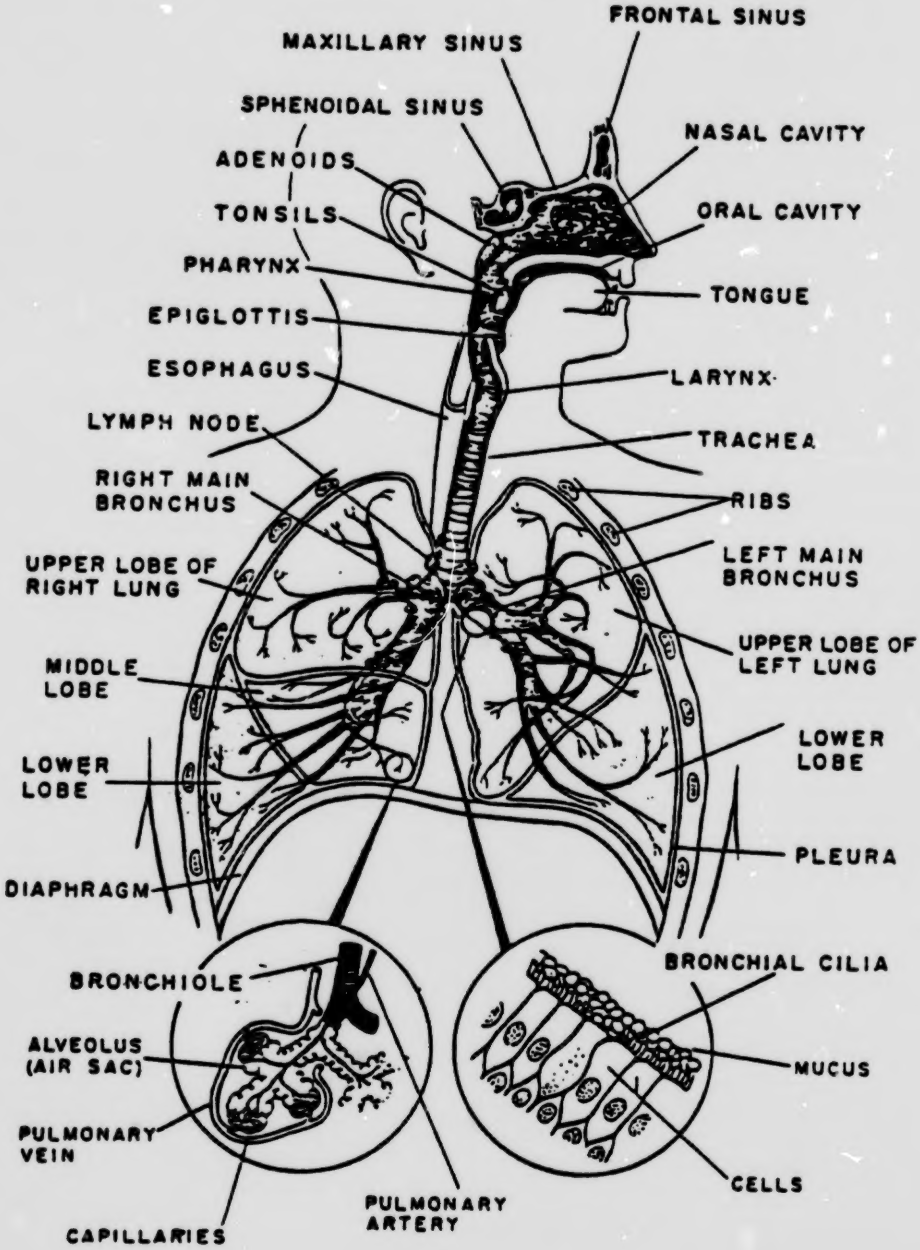
A large vessel carrying blood from the right side of the heart divides into two pulmonary arteries, which enter the lungs and divide repeatedly into smaller arteries and arterioles. The tiny branches, called "capillaries", form a network around the alveoli. The lungs constitute only one part of the overall system supplying oxygen to the tissues. The bloodstream carrying oxygen and the heart, the pump of the transportation system, are clearly of paramount importance. Lungs, bloodstream and heart are thus closely interrelated elements. Disease in any one can cause changes or diseases in the other two. (For example, the scarring of the lungs caused by asbestosis may effect the flow of blood through the lungs and demand the heart to "work harder" to pump the blood. Cor pulmonale, the name given to right heart failure caused by lung disease, may therefore result. Also, reduced oxygen in the blood may signal the body to produce more red blood cells. This in turn may affect the "clottability" of the blood, which may have implications in other diseases.)

For gas exchange to occur the gas has first to be conveyed to the alveoli. This is effected by the bellows function of the lungs, known as ventilation. Once oxygen is in the alveoli it then has to diffuse across the alveolo-capillary membrane before it can be taken up by the blood. The excretion of carbon dioxide depends on the same processes, but in reverse. Unless the capillaries are adequately profused with blood, gas exchange may be impaired. An understanding of respiratory impairment thus comprises an understanding of ventilation, diffusion and profusion; either one, two or all of these may be affected, usually to varying degrees.

Ventilation: Ventilation depends first on the size of the lung (lung volumes), secondly on the calibre of the airways (airways resistance), thirdly on the compliance or elastic properties of the lungs, and lastly on the properties of the chest wall.

Fig. IIIA-1

The Respiratory System


FRONTAL SINUS
MAXILLARY SINUS
SPHENOIDAL SINUS
NASAL CAVITY
ADENOIDS
ORAL CAVITY
TONSILS
PHARYNX
TONGUE
EPIGLOTTIS
ESOPHAGUS
LARYNX
LYMPH NODE
TRACHEA
RIGHT MAIN BRONCHUS
RIBS
UPPER LOBE OF RIGHT LUNG
LEFT MAIN BRONCHUS
UPPER LOBE OF LEFT LUNG
MIDDLE LOBE
LOWER LOBE
LOWER LOBE
PLEURA
DIAPHRAGM
BRONCHIOLE
BRONCHIAL CILIA
ALVEOLUS (AIR SAC)
MUCUS
PULMONARY VEIN
CELLS
PULMONARY ARTERY
CAPILLARIES

Source: American Lung Association

Some aspects of lung volumes can be measured with a spirometer, but others require more complicated apparatus. The total volume of air in the lungs and bronchial tree after a maximum inspiration is known as total lung capacity (TLC). If a person then exhales as much air as he or she possibly can, the volume remaining in the lungs is known as the residual volume (RV), while that which has been expelled is known as the vital capacity (VC) (see Figure IIIA-2). In a healthy young adult the RV is around 20% of the TLC. With increasing age or other reasons contributing to loss of some of the lung's elasticity (as occurs in some occupational lung diseases) the RV increases. The decreased elastic recoil of the lungs is opposed by an unchanged and relatively greater pressure within the pleural space which now maintains the lungs at a higher state of inflation than previously, and the RV constitutes a larger percent of the TLC than prior to loss of elasticity. Tables have been constructed to predict what the VC of an individual of a given age and height should be, and serve as a standard for comparison.

Ventilation depends heavily on the resistance to airflow in the bronchial tree. The forced expiratory volume in one second, (FEV_1), i.e. the amount of air that can be exhaled in one second after a maximum inhalation, usually constitutes approximately 80% of the forced vital capacity. The flow is most rapid as the person begins to exhale, but slows and eventually ceases as the RV is reached. Tables have also been constructed to predict FEV_1 based on age and height.

People with obstructive airways disease (asthma, chronic bronchitis and emphysema) have increased airway resistance and decreased flow rates on pulmonary function testing (i.e. the ratio of FEV_1 to VC is less than 80%). This is known as an "obstructive" ventilatory pattern, (and is depicted in Figure IIIA-2b by a flatter curve).

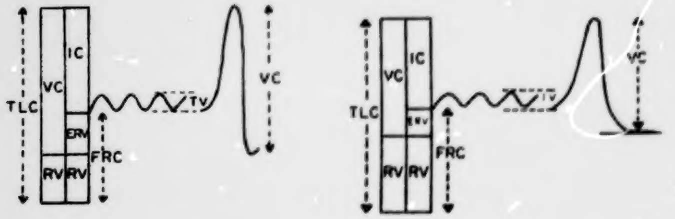
Around 80% of the resistance to airflow in the normal lung is situated in the larger airways, and the resistance of the lower (i.e. smaller) airways is relatively little in comparison. Therefore obstruction in the smaller airway is less likely to be detected by measuring FEV_1. It can be detected by measuring MMEFR (maximum mid-expiratory flow rate), however this is less reproducible and subject to greater error.

Ventilation depends as well on the compliance of the lungs - the measure of their distensibility. The larger and more elastic the lungs, the more compliant. If the lungs become stiff, shrunken and fibriotic (or scarred) as frequently occurs in diseases such as asbestosis and silicosis, the compliance is markedly reduced. An individual with diffuse disease of small airways may have normal FEV_1 and relatively normal airway resistance, but, although the total resistance may be normal, if the resistance in the peripheral airways is increased the lungs are less distensible. When severe enough, this is reflected in a reduction in VC. If the VC is reduced disproportionately to the FEV_1, the defect is described as a "restrictive" ventilatory pattern.

Many industrial lung diseases, particularly the occupation-specific diseases, affect the peripheral parts of the lung due mainly to the physics of particle deposition and to a lesser extent to the clearance mechanisms of the lung. As the lesions are peripherally situated in the smaller airways and alveoli, there is greater difficulty in detecting early abnormalities by conventional lung function tests.

Fig. IIIA-2a

Lung volumes and a spirometric tracing illustrating **vital capacity** in:



i) a **normal** subject

ii) a subject with **"obstructive" airways disease**

iii) a subject with diffusion fibrosis (e.g. asbestosis)-- **"restrictive pattern"**

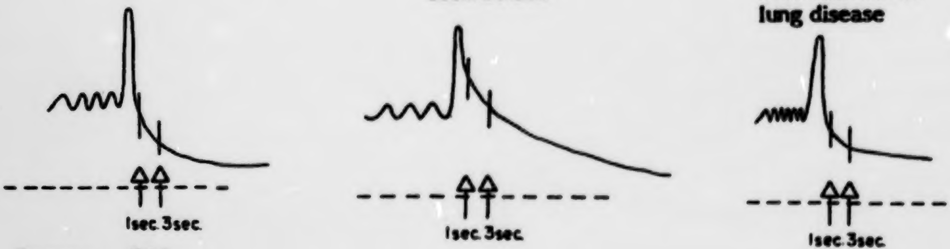
Abbreviations:

TLC - total lung capacity; RV-residual volume; IC-inspiratory capacity; ERV-expiratory reserve volume; VC-vital capacity; TV-tidal volume; FRC-functional residual capacity.

Source: Morgan & Seaton, Occupational Lung Diseases, W.B. Saunders Co., 1975.
Drawn to scale

Fig. IIIA-2b

Forced Expiratory Volume maneuvers in:



i) a **normal subject**

ii) subject with **airways obstruction**

iii) subject with **restrictive lung disease**

Source: Ibid.

Diffusion: Diffusion of gases across the alveolo-capillary membrane depends on the pressure difference between the alveolus and pulmonary artery as well as the state of their surfaces. Although the diffusing capacity of the lungs can be measured, it is influenced by a variety of factors besides the physical state of the alveolo-capillary surface; body size, red blood cell content of the capillaries, and any ventilation-profusion abnormalities will affect the result. The diffusing capacity is reduced in conditions in which the lungs have lost their compliance (asbestosis, beryllosis, etc.), where the alveolar walls are destroyed (in emphysema), in the presence of pleural thickening (characteristic of asbestos exposure), etc. The test of the lung's "diffusing capacity", along with measurements of lung volumes and flow rates, is heavily relied upon in diagnosis and evaluation of respiratory impairment. In the presence of more than one respiratory disease the interpretation of these parameters may be problematic.

Profusion: Maintenance of arterial oxygen and carbon dioxide partial pressures at normal levels requires that the ventilation-profusion ratio of the lungs as a whole remains relatively normal. Although the absolute levels of ventilation and profusion may vary considerably, it is in conditions where mismatching of the two occurs (i.e. ventilation of areas with poor blood flow or profusion of areas that receive minimal ventilation) that the most devastating defects in gas transport take place. Reduction in profusion to areas adequately ventilated occurs when the pulmonary vascular bed is compromised (from a blood clot or pulmonary embolism for example), when the lungs are less compliant than usual, and in emphysema. When the blood crosses capillaries that are adjacent to poorly ventilated alveoli, it also leaves the lung with markedly reduced oxygen saturation and partial pressure. This situation may occur in pneumonia or chronic bronchitis. When severe enough, it results in reduced oxygen available to the tissues of the body and may be evident as cyanosis (the patient appears to have a bluish coloration).

Arterial oxygen desaturation frequently occurs in occupational diseases. This may be due directly to its effect on the diffusion of oxygen across the alveolo-capillary membrane, or due to the fibrosis (the formation of scar tissue) which leads to shrinkage and tearing of tissue and consequent ventilation-profusion abnormalities. Arterial blood gases may also be measured in the diagnosis and assessment of respiratory disease. (As it may be a painful intervention, it is not routinely indicated).

The reduced availability of oxygen may affect the healing potential in other tissues of the body. Moreover, while there is substantial debate regarding whether a person with respiratory disease is more likely to suffer a myocardial infarction (heart attack) than an individual without lung impairment, there is considerable evidence that a person who has a severe lung disease has a greater chance of dying from this event (See Section 3.1.1). These people are also at greater risk of dying from surgery requiring anesthetics. The controversy regarding eligibility for compensation in such cases is therefore understandable.

2.3 *The Deposition and Clearance of Dust from the Lungs*

Dust deposition and retention: When a person takes a breath, air is drawn in through the nose or mouth, down the nasal pharynx and trachea, through the conducting system to the alveoli. The rate of flow of the inspired air decreases as it progresses through the system. Deposition of particles occurs as a consequence of several different physical processes, namely: sedimentation, inertial impaction and diffusion. Particles sediment under the influence of gravity with large particles falling more rapidly. When the particle is being carried along in an air current with changes of direction, the momentum of the particle will cause it to be deposited by impaction, for example at the bifurcation of the bronchial tree. Most inhaled particles are deposited well before the respiratory bronchioles are reached. Nearly all particles greater than 20 microns come in contact with the walls of the bronchi. Most particles below 3 microns reach the respiratory bronchioles and are deposited there, or more commonly, in the alveoli. The gas in the alveoli is turned over at a slower rate than in the conducting system. Therefore the particles which reach the alveoli are more likely to be retained long enough to be deposited.

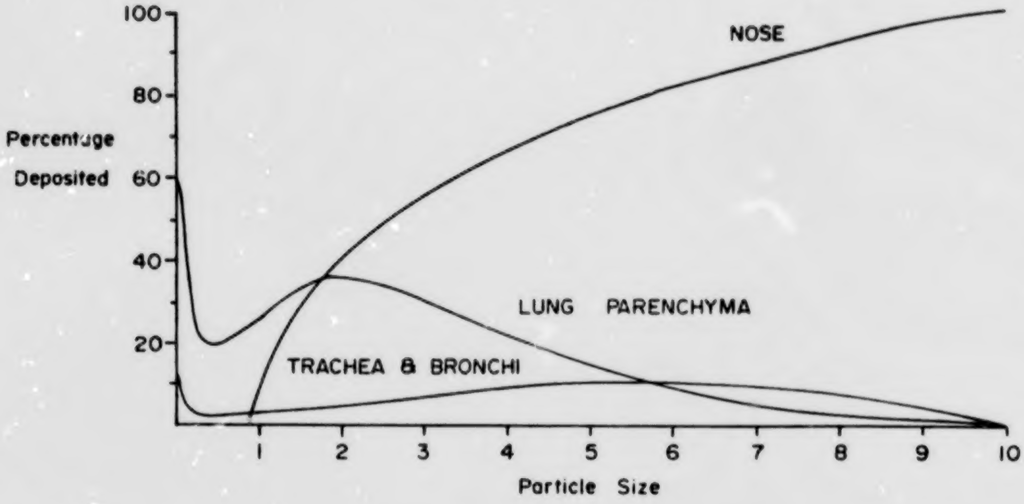
Some particles are deposited not by sedimentation and impaction, but as a result of their random diffusion, (Brownian motion). This mechanism is significant for particles up to 2 microns in diameter and, in fact, is the only mechanism for particles under 0.2 microns. Only elongated particles, such as asbestos, fiberglass and talc may be deposited by interception. Particles over 5 to 10 microns long lodge mostly in the smaller bronchioles, but are capable of some penetration and migration.

Although the likelihood of dust becoming deposited in the lungs is dependent on a large number of factors (including the rate of breathing, the integrity of the clearance mechanism of the lungs and the presence of concomitant disease in the respiratory system), the size of the particles involved is of paramount importance in determining the site of maximum damage. This is represented diagramatically in Figure IIIA-3.

Air quality monitoring devices have been designed making use of these principles. The elutriator, based on a curve designed in Britain, and the midget impinger, designed in the U.S. vary slightly in the dust they sample. Both are designed to sample the amount of dust that would be expected to reach the respiratory bronchioles and alveoli, known as the "respirable fraction". The threshold limit values are, for the most part, designed to reduce disease in this area, (the site of silicosis and asbestosis, for example). Measurements of total inhalable dust are also possible, for which standards exist as well. These standards, however, are often based on the assumption that the total amount of dust constitutes merely a nuisance factor. Less attention has been devoted to measuring the amount of dust that would be expected to potentially damage the airway system and, in fact, no measuring device has been designed specifically for this purpose. There is currently some debate about whether a slightly coarser sample should be developed to focus more on bronchial dust than the present instruments are able to do. This would certainly affect the ability to assess the role of workplace dust in chronic airway disease, a topic of great concern in the workers' compensation system.

Lung Protecting Mechanisms: The air we breathe contains numerous bacteria, dust particles and debris. Depending on their size, these can interfere with the efficient functioning of the lungs in several ways. Occupational dust particles usually act by constricting air passages, reducing elasticity of lung tissue, or thickening or destroying alveolo-

Fig. IIIA-3



Regional deposition of particles as a function of their size in microns. (This assumes a respiratory rate of 15 per minute and a tidal volume of 700 to 750 ml.) Reprinted from Morgan and Seaton, 1975, op.cit. (Based on Health Physics 12, 179, 1966.)

capillary membranes. Lung protecting mechanisms are designed to keep such hazards out of the lungs or remove them as quickly as possible once they gain entry.

The nose constitutes the first line of defence filtering large particles of dust and fibers before they enter the trachial bronchial tree. However, a person doing strenuous work has to resort to mouth breathing to get enough air, thereby bypassing the nasal filtering system completely. Coughing is a protective reflex triggered by dust particles or irritant materials hitting the walls of the trachea or main bronchi. The chest and lungs contract rapidly forcing air out of the lungs quickly and usually expelling foreign bodies or irritants with it.

Those particles deposited in the trachea and bronchial tree are removed by the "mucociliary escalator". This continuous clearing mechanism extends from the terminal bronchioles (just proximal to the respiratory bronchioles) right up to the glottis. Basically, special secretory cells keep the lining of these passages coated with a thin layer of watery mucous which traps dust particles and micro organisms. The airways are also lined with cells that have moving hairlike extensions (called "cilia") that stroke upward sweeping the mucous continuously up the airways. When the mucous reaches the trachea, it is swallowed, usually subconsciously, or spat out. The swallowing of inhaled debris in this manner explains how the gastro-intestinal system is affected by certain occupational hazards (e.g. asbestos). Airway obstruction as seen in bronchitis increases the fraction of particles trapped in the larger airways. Also the beating of cilia and hence the effectiveness of this clearing mechanism is also influenced by chemicals in the environment (ionic changes and oxidants) and cigarette smoke.

Both cilia and mucous secretory cells are few or absent in the respiratory bronchioles and alveoli. Dust particles, debris and cells must be transported either to the trachial-bronchial mucociliary escalator or into the specialized drainage channels, the lymphatics, which surround the blood vessels and bronchi. Knowledge about the alveolar transportation system is fragmentary. Alveolar macrophage are highly specialized mobile cells that engulf and digest bacteria and viruses. In the process the macrophage themselves may be destroyed and the resulting debris is swept away by the mucociliary escalator. Macrophage also engulf and attempt to digest such lung hazards as dusts. When toxic dust is involved, the macrophage are also destroyed and the dust often remains in the lungs. Experimental evidence shows that the overall clearing of this area is slower than of the larger airways, usually taking days rather than hours and accounts, at least in part, for the increased effects seen in this area by certain occupational hazards.

Thus dust of different particle size causes maximum damage at different locations in the lungs. Sampling techniques concentrate heavily on the fraction of dust that is expected to reach the respiratory bronchioles and alveoli, and fail to properly measure dust that may damage the airways. Although the lung has clearance mechanisms to protect itself from disease, a certain percentage of dust gets in regardless; the greater the exposure, the greater the amount. In the presence of other substances such as chemicals or cigarette smoke, damage to the clearance mechanisms may further decrease their effectiveness, and consequently even shift the pattern of damage. Appreciating the many factors that affect dust deposition and retention helps explain the extreme variability in clinical manifestation of dust-induced disease. It also helps to illustrate how the presence of some factors affects the "susceptibility" to other factors (e.g. toxic substances or cigarette smoking may affect the susceptibility to infection).

2.4 *Pathological Reactions of the Lung*

The effect on the lung of the various pollutants is also largely dependent on the biological activity of the material encountered. The inhalation of vapours and dust encountered occupationally may produce almost the full range of possible lung responses. The inhaled material can behave as an acute irritant evoking an acute non-specific inflammatory reaction; or it could be a low-grade irritant, evoking a low-grade non-specific chronic response; or when combined with or containing a protein, it may elicit a variety of hyper-sensitive or "allergic" lung reactions.

Hazards that are similar in physical form may vary considerably in their disease-causing powers. Some mineral dusts are thought to be "merely a nuisance"; other mineral dusts irritate lung tissue and cause acute inflammation, some can lead to chronic obstructive pulmonary disease; others induce fibrosis or even cancer. Some gases suffocate; others poison body systems; still others produce chemical burns in the lungs and other tissues.

The ability of individuals to handle hazards and to resist disease varies with age, smoking habits, and perhaps other less-understood factors such as nutritional status and stress. Individual variability certainly will modify the consequences of exposure. Anatomical variations, for example, of lymphatic drainage, may also alter the site of dust retention in different individuals. Moreover, exposure in an industrial workplace is seldom as pure as that in a designed laboratory experiment. It is not surprising therefore that particular presentations of respiratory diseases in specific individuals are varied and complex. Nevertheless, it is convenient to divide occupational lung diseases into various categories to illustrate briefly the variety of pathological reactions that may occur and to allude briefly to some of the issues that may arise with respect to compensation of the diseases in each category. (See Figure IIIA-4.)

Chemical Pneumonitis (Inflammation of the Lungs): After acute exposure to certain chemicals, the lung may become edematous (swollen), hemorrhagic (bleeding) and inflamed, with various degrees of induration (hardness). (If exposure is sufficiently intense, it may be fatal.) Similar changes may also occur in the larynx, trachea and bronchi, causing laryngitis, tracheitis and bronchitis respectively. If the substance is gaseous, the more soluble it is, the higher up in the respiratory tree is the area of greatest damage. Thus, highly soluble gases such as chlorine, ammonia and sulfur dioxide (So_2) tend to dissolve in the lining of the upper airways to produce a tracheobronchitis. In contrast, less soluble gases such as nitrogen dioxide (No_2) and phosgene tend to penetrate further and cause a bronchiolitis or alveolitis. The intensity of the response in this category of lung disease depends on the nature of the noxious material and on the dosage and duration of exposure.

Early in the reaction to substances such as nitrous fumes (for example in silo-fillers' diseases) or metal fumes and gases produced for example in

Fig. IIIA-4

Spectrum of Respiratory Pathology (Simplified)

Toxic or Inflamatory Reactions	**Hypersensitivity Reactions**	**Fibrotic Reactions** (Scarring)	**Malignant Transformation**
(such as tracheitis, bronchitis, bronchiolitis, alveolitis, etc.)	(such as asthma, allergic alveolitis, etc.)	(such as silicosis, coal workers' pneumoconiosis, etc.)	(cancer)
Caused by	**Caused by**	**Caused by**	**Caused by**
e.g. chlorine ammonia SO_2 NO_2 phosgene etc.	e.g. animal dander enzyme detergents trimellitic anbydride toluene diisocynates wood dusts mouldy hay mushroom compost etc.	e.g. organic dusts inorganic dusts silica silicates others (See Appendix 2)	See Chapter II

welding operations, a non-specific inflammation occurs. Later, fibrous (non-elastic tissue) and cellular components accumulate, the tissue may become destroyed (necrotic) and slough off. Possible sequelae include the obliteration of the bronchiole by the effect of the inflammation (bronchiolitis obliterans), intra-alveolar and interstitial fibrosis (non-elastic tissue formation within the alveoli or within the support structure of the cells) or prominent emphysema (destruction of the alveolar walls). Some of the latter sequelae may occur in a patient without a history of a dramatic illness following acute exposure. Exposure may be minimal over a long period with lung lesions developing insidiously.

Although the acute illnesses often are able to be linked to the workplace, at present it is very difficult to ascertain the role of small but repeated exposures. Threshold limits of exposure, if they have been set, are usually aimed at preventing the acute reaction only. Not only can low levels of exposure to NO_2, SO_2 and ozone, for example, produce toxic effects in their own right, but they may also cause an increase in the nonspecific sensitivity of the airways to other substances which may be harmless under normal conditions.

Hypersensitivity Occupational Lung Disease: Several occupational lung diseases operate through hypersensitivity mechanisms. Certain substances that contain proteins or are capable of combining with protein may act as "antigens" stimulating the body to mount an immunological reaction to them in a manner that is out of proportion or harmful to the body. In contrast to the inflammatory reactions described above which affect virtually all exposed workers, only a certain, usually small proportion, of people will develop a hypersensitivity, or allergy, to a particular substance. Although an over-simplification, four distinct types of hypersensitivity reactions have been recognized: Type I, or immediate hypersensitivity, occurs when an immunological molecule called IgE located on the surface of certain body cells (mast cells or basophils) binds with its correspondent antigen to cause the release of histamine and other active substances from cells. The period of sensitization may last from several months to several years, depending on many factors including level of exposure. Certain individuals appear to be genetically predisposed to Type I reactions and are referred to as "atopic". Type II responses occur when antibodies circulating in the blood are directed against components of cell surfaces, causing damage to the cell. Type III reactions occur when antibodies of the IgG type combine with antigens to form small precipitating complexes which interact with a series of proteins called complements and are ingested and enzymatically digested by cells called phagocytes. These enzymes may leak out to damage surrounding tissue. Type IV or delayed hypersensitivity is maximal at 48 to 72 hours after exposure and is mediated to a large extent by a type of white blood cell called a lymphocyte which has already been sensitized to the antigen. Genetic predispositions to Type I reactions have no bearing on the possible development of other types of hypersensitivity response. This is important in occupational lung diseases. Also, sensitivity to some substances does not necessarily imply increased likelihood of becoming sensitive to others.

Occupational asthma is a hypersensitivity reaction which occurs mainly in the small bronchi and bronchioles. It is characterized by spasm of the muscles in the airway walls (bronchospasm) which narrows the airways, causing the victim to wheeze as he or she struggles to breathe. A thick sticky mucous (containing characteristic cells particularly eosinophils) is secreted and is difficult to clear from the airways. If this feature becomes predominant, the asthmatic condition may be initially mistaken for chronic bronchitis. The thickening of the walls and overdevelopment of the mucous secreting apparatus is a feature actually common to both diseases.

Often the asthmatic reaction is not limited to the immediate Type I reaction, but also involves bronchial constriction 4 to 10 hours after exposure, possibly reflecting a Type III component. The occupational asthma that occurs in cereal workers, wood-workers, printers or workers exposed to enzymatic detergents, isocyanates, various metals and other substances has been recognized. The list of occupational allergens has been growing extremely rapidly in recent years. As the clinical features are not always characteristic and difficulties are often encountered in identifying the causative agent, establishing compensability of the condition is also often problematic.

Allergic alveolitis, as the name implies, involves an allergic inflammation affecting the alveolar wall. It also usually affects the surrounding tissue, including the bronchioles and pulmonary arterioles, and is often caused by fine organic dusts that are inhaled into these areas. (For example, farmers' lung, grain handlers' disease and mushroom-workers' disease all fall into this category). Some of these diseases may easily be confused with non-occupational diseases, for example, "idiopathic" fibrosing alveolitis for which the cause is not known.

The alveolitis episodes usually begin several hours after exposure to the causative hazard. The mechanism is largely unknown but may represent either a Type III or a Type IV reaction. The alveolar walls become swollen and the distribution of the various types of white cells is disturbed. Cough, fever and difficulty breathing may last several days and can be quite serious or life-threatening. Moreover, frequent episodes, especially if they are mild enough to go unnoticed and untreated, sometimes lead to a diffuse fibrosis (scarring) of the alveolar-walls, restricting the functioning of the lungs with all its concomitant effects. Occasionally a single severe episode may lead to extensive fibrosis, although this is less common. A slowly continuing hypersensitivity inflammation is postulated. The pattern of fibrosis may vary from fine fibrosis to the development of emphysematous sacs surrounding areas of fibrosis. Some patients may even proceed from a single attack to prominent emphysema.

Although not included in the schedule of diseases, a variety of cases of this variety have been compensated in Ontario. However, unless the situation of exposure has been well-studied, problems in recognizing the work-relatedness of some of these conditions may also occur.

Pulmonary Fibrosis and Variations: The reaction of the lungs to inorganic dusts depends not only on the size of the particles, but on the intensity and length of exposure and on the nature of the dust inhaled. One of several types of reactions may occur when dust particles penetrate to the respiratory bronchioles or alveoli. The macrophage can engulf certain kinds of dust in large amounts without showing toxic effects (for example, tin, iron and pure carbon). In this situation, the macrophage may accumulate in alveoli and are held there by a few fibroblasts (cells which produce a web of fibres).

When the dust is asbestos, the lesions consist initially of deposition of fibres, machophage and debris; later the web in which these deposits are enmeshed becomes coarsened and replaced by inelastic collagen fibres. Initially these reactions are confined to discrete individual respiratory bronchioles scattered throughout the lungs. After further exposure, more and more respiratory bronchioles become involved, then all the respiratory bronchioles arising from specific terminal bronchioles become thickened, and eventually the alveoli are involved. The individual lesions coalesce and the dense fibrous network becomes increasingly dense causing the lung to become stiffened and inelastic, and destroying the air spaces. The alveolo-capillary membrane may also thicken, decreasing the diffusing capacity of gases through it. Once sufficient asbestos dust has been retained, the lesions will progress even if no further exposure occurs. (This will be alluded to again below as it is particularly important from the compensation viewpoint.) The fibrous dust has a tendency to drift toward the pleura, the lining of the lungs. The pleura becomes fibrotic (scarred) and calcium deposits gradually accumulate. The clinical symptoms and natural history of the disease will be discussed more fully below. The increasing breathlessness and disability that the patient experiences in asbestosis is essentially due to diffuse interstitial fibrosis which produces a "small tight lung" and all its concomitant effects on ventilation, diffusion and perfusion.

Silica quartz, or crystalline free silica, may produce a variety of different patterns of reaction. In classical silicosis the dust is ingested by the macrophage causing the enzymes within the cells to be released, killing the cell and leaking out into the alveoli. The quartz is then taken up by additional macrophage and the process repeats itself with more and more fibroblasts and macrophage attracted to the area. Nodules of these cells form initially around terminal bronchioles and proximal respiratory bronchioles. The arteriole supplying the respiratory bronchioles and the lymphatic channels are involved. The silicotic nodules also continue to proliferate in both lung tissue and lymph glands after exposure has ceased. The classic symptom here too is "dificulty breathing" as will be outlined in Section 3.1.1.

Whereas in asbestosis the fibrosis is diffuse, in classical silicosis the nodules are discrete. This may explain why silicosis is less disabling in its early stages. Later, the nodules may coalesce to form conglomerate lesions which may occupy large areas of the lung and cause significant disability. The pattern of fibrosis in coal workers' pneumoconiosis represents still a different picture and may be associated with emphysema surrounding the fibrosis. However, other patterns of silicosis also occur. For example, a diffuse interstitial fibrosis similar to asbestosis may occur, and if the exposure is intense and acute a very different disease pattern may result which may even be associated with kidney failure.

Thus, there are several different reactions of the lung to chronic exposure to mineral dust. The first type, high dust accumulation in the lungs, but little scarring, is characteristic of exposure to iron, tin, barium and early coal workers' pneumoconiosis; high dust accumulation with massive pulmonary fibrosis may characterize more severe coal workers' pneumoconiosis; relatively little dust content but generalized nodular fibrosis is the hallmark of classic silicosis, and pneumoconiosis characterized by diffuse pulmonary fibrosis is seen in asbestosis and berylliosis. Fibrosis also occurs in response to organic dust as mentioned earlier.

It must be remembered that free silica and silicates constitute a large part of the earth's crust. In most rocks free silica comprises only a fraction of the total material. A very large proportion, however, may be comprised of combined silica or silicates, (formed by a combination of various cations such as aluminum, calcium, iron, magnesium or potassium into the silica crystal). The disease potential of the silicates is variable, but in general is much less than that of free silica, with the notable exception of the fibrous silicates of the asbestos group. Some authors feel that the phrase "mixed dust pneumoconiosis" is uninformative, since under most circumstances all dusts are to some degree mixed. It must, therefore, be stressed that not only are the clinical presentations caused by the separate agents rarely pure, but workers are often exposed to a variety of different types of dust. These factors contribute to the often noted imprecision of diagnosis.

(Aside from the acute or chronic inflammatory reactions, the hypersensitivity reactions, and the fibrotic reactions, lung tissue may also respond to occupational hazards by undergoing malignant transformation. There are several different types of lung cancer that may develop, all of which are devastating, but will not be discussed in this chapter as the compensation issues presented by the latter category were discussed at length in Chapter II.)

The reader can now appreciate that respiratory impairment may be caused by reduction in the ventilation, diffusion or perfusion functions of the lung or a combination thereof; particles of different size with different toxicological properties may affect the lungs in different ways; neither are the pathological entities mutually exclusive nor are occupational exposures in the real world mainly to pure single agents. It should also be apparent from the above that the pathology caused by work-related respiratory disease can be quite varied and can considerably overlap with non-work-related diseases. Thus a major issue, if not "the major issue" in compensation of respiratory diseases revolves around the determination of work-relatedness. It should be noted, however, that although the pathology and pathophysiology of the diseases differ, the symptoms experienced by the patient are often remarkably similar. Indeed the major feature of all these diseases is breathlessness, and, at the risk of being overly simplistic, in all these diseases the worker may first notice shortness of breath on exertion and occasional cough, gradually the ability to do things may seem reduced, infections may be more frequent and more severe, and finally breathing dificulties may totally confine the patient to bed. The next section will address these issues as they relate to some specific occupational respiratory diseases encountered in Ontario.

3. The Nature and Prevalence of Occupational Lung Diseases

3.1 *Pneumoconiosis*

3.1.1 *The Pathogenesis, Clinical Presentation, and Natural History of Pneumoconioses Prevalent in Ontario*

Pneumoconiosis refers to the accumulation of inorganic dust in the lungs, with or without non-neoplastic tissue reaction to its presence. These diseases are essentially specific to the occupational agent. Although still subject to controversy with respect to many compensation matters, the issue of causation is more straight forward, compared to the non-specific lung diseases, such as bronchitis or emphysema.

In March, 1975, the President of The Canadian Thoracic Society expressed concern over the lack of uniformity amongst the provinces in relation to the diagnosis and classification of pneumoconiosis, disability assessments and the amount of pensions awarded. A task force consisting of nominees endorsed by the Canadian Medical Association, L'Associasion des Medecins de Langue Francais du Canada and the Royal College of Physicians and Surgeons of Canada, was consequently set up to establish a classification of pneumoconiosis, to develop criteria for the diagnosis of asbestosis, silicosis and coal workers' pneumoconiosis and to develop criteria for assessment of disability due to these three pneumoconioses. Their conclusions and recommendations will be discussed in subsequent sections as relevant.

An etiological classification of pneumoconiosis adapted from that proposed by this Task Force on Respiratory Diseases is presented in Appendix 2. For purposes of this report, the specific characteristics of only those occupational lung diseases which are common in Ontario will be summarized (for example, coal workers' pneumoconiosis, important to the United States and elsewhere in Canada will not be discussed here). Although future studies may reveal the prevalence of yet unrecognized problems, silicosis and asbestosis remain the most important of the pneumoconioses in this jurisdiction.

Silicosis: Silicosis is a disease of the lung characterized by nodules of scar tissue (hyaline nodular fibrosis) produced by prolonged inhalation of free silica dust. (The acute form of silicosis may result in less scarring and more of an exudative reaction from the oozing of fluids from the damaged cells and, therefore, has a somewhat different clinical presentation.) The theories as to how silica produces this lung damage are varied as summarized by the Task Force and include (a) a mechanical theory that the sharp edges of the particles injure the tissues and stimulate the scarring, (b) an electrical theory that silica crystals injure the fibroblasts by an electrical discharge when they are compressed, (c) a chemical theory that hydrogen bonding takes place between the silica particles and the phagosomes causing the latter to rupture and stimulate fibrosis and (d) an auto-immune theory that the silica triggers the host defence to react against normal tissue to produce fibrosis[14]. (Although the typical immunological reaction does not occur, there is an increased incidence of auto-immune disorders such as scleroderma, rheumatoid arthritis or systemic lupus erythematosis in silicotics which have lead some to this hypothesis).[15]

In any case, workers exposed to silica in mining, quarrying and tunnelling, in stone cutting, dressing, polishing, and cleaning monumental masonry, in the production of abrasives, abrasive blasting and gravel crushing, in glass manufacturing, foundry work, boiler scaling, vitreous enamelling or the manufacture of pottery, porcelain and lining bricks may all develop this disease.[16]

The Task Force divided silicosis into three forms according to duration of dust exposure as follows: chronic silicosis where the duration of exposure to silica is usually in excess of 20 years, accelerated silicosis where the duration of exposure is usually between 5 and 20 years, and acute silicosis where the duration of exposure is shorter than 5 years.

Silicosis is often first recognized by an increase in the "lung markings", i.e. increased lace-like densities on the chest x-ray. These changes may not be evident until years after the exposed person has left the workplace. This is usually detected first in the upper halves of each lung and is associated with a blurring of the hila. It is at this stage that there is the greatest doubt as to whether the chest film is normal or not. Later rounded opacities of 1 to 10 millimetres in diameter become more clearly delineated, constituting the hallmark of the disease. The nodules may coalesce to form opacities greater than 10 millimetres in diameter, thus constituting "conglomerate silicosis". These lesions may be associated with adjacent hyperlucent areas, due to the presence of compensatory emphysema. The lesions may now appear more prevalent around the hila and may cavitate. "Egg shell" calcifications of lymph nodes in the same area may be evident. When cavitation occurs it may be associated with infection by the tuberculosis organism. This T.B. may develop insidiously and be difficult to detect radiologically. Involvement of the pleura rarely occurs in silicosis.[17] (In acute silicosis the chest x-ray becomes abnormal much more quickly and its basic feature is a reticulo-nodular pattern producing a hazy lacy chest x-ray picture.)

The x-ray features of silicosis have never been reported to regress, as the disease never really improves. Abnormalities in routine lung function tests do not usually occur until well after the x-ray features are present, however, damage to some extent may well have taken place. By the time conglomerate silicosis is present, there is usually gross evidence of lung restriction with reduced total lung capacity and vital capacity. (In acute silicosis the restrictive defect progresses rapidly with shrinking lung volumes and disturbed gas exchange.)

Pathological examination will usually reveal silicotic nodules and fibrosis with the silica concentrated in the alveolar walls and macrophage. Lung biopsy however is not indicated routinely and it is recognized that the tissue obtained might not always be diagnostic.[18] (This is important from the compensation point of view and will be alluded to in the next chapter).

It is now well established that simple silicosis by itself is usually associated with few symptoms. The cough, sputum and breathlessness that many silicotic workers develop is attributed to chronic bronchitis which often accompanies silicosis. The most important symptom of silicosis is shortness of breath on exertion which usually occurs when the x-ray changes of complicated rather than merely simple silicosis have developed. Evidence of fibrosis on physical examination is usually absent until the late stage. Then, even in the absence of chronic bronchitis, prolongation of forced expiratory time may occur indicating airway obstruction resulting from the loss of retractive forces in the lungs related to compensatory emphysema (i.e. destruction of air sac walls surrounding the scarring).

The fact that silicosis is frequently accompanied by chronic bronchitis is well documented. However, some contend that this occurs only in silicotics who smoke. Consequently, the complication of tuberculosis in silicosis is considered compensable while the "complication" of chronic bronchitis is not. However, distinguishing the respiratory impairment attributable to silicosis from the respiratory impairment attributable to chronic obstructive lung disease caused by smoking is not at all clear cut. The reasons for this are twofold: first, it has been shown that dusty environments, often the same environments which can cause pneumoconiosis, can also cause airway obstruction. (This will be discussed in the next section.) Secondly, the existence of airway obstruction in an individual with pneumoconiosis cannot simply be written off to smoking, because, as the disease progresses, the fibrosis around the bronchi or the presence of complications including infection themselves can cause a superimposed airway obstruction. Thus the identification of a pattern of abnormal pulmonary function attributable to silicosis may be relatively easy when the syndrome is fully developed in the intermediate stages of the condition. However, it is more difficult in the early stages of the diseases when the findings may lie within the limits of normal variation, as well as in the late stage, when the pattern may be obscured by the development of secondary changes due to chronic non-specific lung disease with an obstructive pattern predominating. In light of the limitations in the accuracy of diagnosis and assessment techniques, (as will be discussed in Section 4) the temptation to attribute "obstructive lung disease" to smoking and only acknowledge the restrictive component as work-related is not justifiable. The essence here is that not only is there no clear-cut distinction between obstructive and restrictive lung diseases, as explained in Section 2.2, but silicosis itself is neither purely one nor the other.

The progression of the disease depends not only on the intensity of dust exposure and the resulting fibrosis (and possible airway obstruction) but also on concomitant disease, especially the presence of pulmonary infection, such as tuberculosis. However, simple silicosis has a definite tendency to progress even in the absence of T.B., whether or not the subject remains exposed to dust.[19] The same is true once the complicated disease has developed and even if the masses do not increase in size, disability may continue to increase relating to retraction of the rest of the lung.[20] For the patient this means increasing shortness of breath, sometimes bothersome cough and chest discomfort. Moreover, it means a gradually decreasing ability to perform his or her job, decreasing exercise tolerance which restricts leisure activities, and possibly eventual confinement to home or even bed. It is widely known that complicated silicosis may lead to chronic respiratory failure (possibly requiring the use of oxygen on occasion, and leading to cor pulmonale with its possible consequences on general cardiac function). The frequency with which these complications occur may vary from industry to industry. It has indeed been shown (as will be discussed in section 3.1.2) that silicotics in Ontario have a higher all-cause mortality rate largely attributable to increased death rate from respiratory diseases compared to the general population.[21]

The Task Force recommended that any person who shows evidence of silicosis after less than ten years of exposure should be removed from exposure.[22] They concluded that it is not known at what radiological stage of *simple* silicosis all workers should be removed from silica exposure, however, they recommended that all workers with *conglomerate* silicosis should be removed. Other experts, such as Anthony Seaton, urge that once simple silicosis has developed, it is undesirable that the worker continue to be exposed to the dust, as this will increase the likelihood of massive fibrosis occurring.[23] Seaton does, however, acknowledge that this advice may be modified in view of the fact that most cases of silicosis occur towards the end of a man's working life. This matter is understandably controversial, especially in that it has important implications with respect to compensation.

Another major point of contention is the relationship between silicosis and cardiac death. Although physiologically one would expect that the added strain on the heart caused by decreasing oxygenation that may be caused by silicosis would contribute to increased cardiac mortality, there is little epidemiological evidence to substantiate this premise. Silicotics in Ontario do not have an increased risk of ischemic heart disease[24], and several studies of coal miners failed to show an increased mortality from cardiac disease.[25] These studies, however, did not specifically look at those with severe pneumoconisis, and, in fact, the latter group of studies did not differentiate between coal miners with pneumoconiosis and those without. Some experts have claimed that because chronic hypoxia (oxygen lack) causes dilation of the coronary arteries, coronary blood flow is actually improved in these people, decreasing their likelihood of an infarct.[26] They consequently conclude that chronic lung disease is unlikely to contribute to the development of cardiac death. However, hypoxia has been shown to increase the extent and severity of injury in experimentally-induced coronary artery occlusion in dogs[27], and it is now known that hypoxia reduces overall cardiac function in humans: aggravating heart failure, increasing electrocardiographic abnormalities and increasing the risk of severe arrhythmias (possibly fatal disorders in the heart rhythm).[28] Numerous studies have shown that cardiac arrhythmias are common in patients with chronic lung disease, especially during exacerbations of their respiratory disease[29]. Mechanisms explaining these observations on theoretical grounds were, in fact postulated over 25 years ago[30] Furthermore, experts have pointed out that hypoxia is not the only factor promoting arrhythmia in patients with chronic lung disease. Senior and his colleagues[31] stress that pulmonary hypertension and drugs such as digitalis, used in the treatment of right heart failure, should not be overlooked as possible causes of arrhythmia. They also note that atrial arrhythmias are more common in patients with chronic lung disease than in patients with coronary artery disease, and the incidence of ventricular arrhythmias is about the same in the two conditions[31]. Moreover, several studies have even suggested that diminished pulmonary vital capacity may be a risk factor in the development of myocardial infarction and sudden cardiac death, although its "causal" as opposed to "contributory" role could not be determined.[32] Some experts, such as Davies[33], have asserted that the hypoxia associated with complicated pneumoconiosis can surely increase the risk of dying from myocardial infarction and therefore cardiac death in workers with severe pneumoconiosis should be compensable. (This issue is discussed in Chapter IV with respect to the eligibility for fatal benefits of survivors of workers who suffered from occupational lung disease.)

In summary, workers exposed to silica seem to suffer more from chronic bronchitis than non-exposed workers. When the effects of chronic bronchitis on ventilatory function are excluded, simple silicosis appears to cause little impairment other than possibly some oxygen desaturation on exertion in the later stages. However, quite often silicotics do have chronic obstructive lung disease and eliminating the influence of the latter is not an easy task. Complicated silicosis may be associated with considerable impairment manifested by decreasing lung volumes, impaired gas diffusion and exercise intolerence. The disease is progressive sometimes even in the absence of continued exposures and the issue of criteria to remove a worker from exposure is hotly debated. Needless to say, the goal is to prevent the disease through the institution of engineering controls to reduce or eliminate exposure from the workplace rather than eliminating workers from this hazard. Lastly, silicotics may suffer considerable disability and have an elevated all-cause mortality rate largely attributable to increased death rate from respiratory disease. Although the stress of the disease may contribute to the worker's ultimate demise from ischemic heart disease, there is little evidence that the physiological disorders produced by silicosis itself contribute directly to coronary artery disease or to the likelihood of infarction. The possibility of increased risk of fatal cardiac arhythmias remains controversial, but there seems to be a consensus that serious lung disease is associated with worse prognosis in the event of a myocudial infaction.

Asbestosis: Asbestosis is a disease of the lung characterized by diffuse interstitial fibrosis produced by the prolonged inhalation of respirable asbestos fibres. It may or may not be accompanied by reaction of the pleura. Asbestos exposure can also lead to development of abnormalities in other organs, some of which were discussed in the chapter on malignancy.

Asbestos refers to a group of fibrous silicates, however, there is no clear indication that the different types of asbestos have different biological effects.[35] An understanding of how asbestos dust produces disease is even less clear than for silica and again, mechanical theories, chemical theories and auto-immune theories have been suggested. It is known, however, that the length of the fibres and more importantly, their aerodynamic characteristics influence the penetration and site of deposition in the lungs.

Exposure to asbestos is ubiquitous. A list of occupations at risk prepared by Dr. G. Gibbs and cited by Becklake and the Task Force on Occupational Respiratory Diseases (Pneumonconioses) is included in Appendix 3a.

A dose-response relationship has been put forward with the dose expressed as the product of average dust concentration times the duration of exposure. This approach itself has been criticized, as factors such as the rate of exposure are not taken into account. The standards of "acceptable" exposure vary from country to country and even from province to province in Canada.

The first indication in a worker exposed to asbestos that the disease may be present is often shortness of breath on exertion. This may be slight at first but may increase until the shortness of breath is even present at rest. The symptoms may develop insidiously seldom becoming manifest within 10 years of first exposure. It may also progress even after the worker is withdrawn from exposure. Frequently a cough is noted, which may be dry or productive of sputum. The physical examination may reveal crackles in the lower portions of the lungs. These "rales" as they are called can be described as fine, dry, high-pitched and do not clear with coughing. In the early stages, they are best heard at the end of a deep expiration at both lung bases and are indistinguishable from those heard in other forms of pulmonary fibrosis and even occasionally in pneumonia or pulmonary edema. Unlike in silicosis, these signs may be present early in the disease, possibly preceding radiological changes, and they become more widespread as the disease progresses. In the more advanced stages, cyanosis may be detectable depicting insufficient oxygenation, and signs of right heart failure may be present.

The first radiological changes noted in asbestosis consist of a network of fine irregularly-shaped linear densities usually more prevalent in the lower zones of the lungs. These become more numerous and coarser as the disease progresses. Eventually, the normal structures are obliterated, signs of cardiac decomposition develop and the x-ray is characterized by a "honey-combing" appearance. However, progression towards massive lesions as occurs in silicosis is rarely observed in asbestosis, and the lymph nodes rarely become visibly enlarged. Involvement of the pleura is evident in a majority of cases and is characteristic of asbestos effect. The x-ray may show pleural thickening, calcification of plaques and blunting of the costophrenic angles (angle between the ribs and diaphragm making up the lower side corners of the x-ray). Radiological diagnosis of the early stages of asbestosis is difficult and there is considerable inter-observer variability[36] as will be discussed below.

Also unlike silicosis, abnormalities in pulmonary function tests may be evident before radiological changes are noted.[37] Asbestosis produces the classical picture of the restrictive disease: increased lung recoil and small lung volumes (decreased vital capacity, decreased total lung capacity and slightly increased residual volume) as well as the reduced diffusing capacity, which some authors claim relates more closely to the degree of impairment from asbestosis than do the other tests.[38] Exercise intolerance and oxygen desaturation on exercise may precede these findings and other authors claim that increase in exercise ventilation and decreased vital capacity are the most sensitive indicators of the disease.[39] Detection of asbestosis thus depends on clinical examination, x-ray findings and lung function tests, all three of which have at times been claimed by various authorities to be the most reliable in early diagnosis. It is generally agreed that asbestosis is a difficult disease to diagnose; ironically, some authors feel that it is actually overdiagnosed.[40]

In terms of pathological findings, as well, asbestosis is not significantly different from the non-nodular interstitial fibrosis of non-occupational origin. There is disagreement as to whether the lesions first appear at the level of the respiratory bronchioles or the alveolar sacs. Nevertheless, lung biopsies (other than at autopsy, of course) are not normally indicated for diagnostic or compensation purposes due to the risks and uncertainties involved. Also, the presence of asbestos bodies in the lungs is indicative only of asbestos exposure, and does not offer proof of disease.

Tuberculosis is not frequently associated with asbestosis, the main complications of which are respiratory failure and cor pulmonale, which according to Seaton usually occur within 15 years of the onset of the disease.[41] Cough, sputum and weight loss frequently occur as the patient is subject to frequent respiratory infections. These respiratory infections, in turn, may trigger episodes of respiratory failure, in which the oxygen concentration falls and carbon dioxide is retained. The patient may become confused, sleep is disturbed and the cough reflex becomes less effective causing more sputum to be retained. Respiratory function then deteriorates further, resulting in severe lack of oxygen and excess of carbon dioxide, and in turn, further mental deterioration. Death may occur if untreated or if mistakenly diagnosed as primary cardiac disease and the patient is inappropriately sedated resulting in further oxygen deprivation.

As is the case with silicosis, the disease tends to progress slowly. According to the Task Force, however, asbestosis may show some accelerated evolution after a period of slow progression.[42] Although it is not known exactly what percentage of asbestotics follow the pattern of events described above, it is known that their overall mortality is definitely increased, as is their mortality from non-malignant respiratory disease as well as death from malignancies.

The evidence that withdrawing the subject from exposure will halt or slow the progression of asbestosis is controversial. The Task Force recommends that the worker be removed from asbestos-dust exposure when the diagnosis is established.[43] Again the goal, of course, is to prevent the disease completely through elimination of exposure. The fact that asbestos is a carcinogen militates even more strongly for its very strict control through industrial hygiene practices. No doubt, the Dupre Commission will have much to say in this regard.

In summary, asbestosis is a difficult disease to diagnose and may be present even in the absence of x-ray evidence. It is certainly more disabling than simple silicosis and definitely affects mortality as well as morbidity. The issue of relocation programs is complicated by the fact that asbestos is a carcinogen.

3.1.2 *Studies on the Prevalence and Severity of Pneumoconioses in Ontario*

By the 1960s concern over health and safety conditions was becoming increasingly vocal. The McGillivary Report on Workmen's Compensation in Ontario in 1967 addressed some of the issues and outlined the need for better documentation of the extent of respiratory hazards in Ontario workplaces.[44] Since that time a series of studies has been undertaken on various respiratory conditions.

Despite the recognition of the problem, the current reality is that neither the size of the population at risk nor the incidence and prevalence of pneumoconiosis could yet be estimated with any degree of certainty. Evidence suggests that both the incidence and severity of silicosis, the most common of the pneumoconioses in Ontario, seems to be declining although it is still a persistent problem. Ontario-based studies have once again shown that asbestosis increases the mortality as well as the morbidity of its victims, and a recent Ontario study has shown that silicosis is also not a benign disease. The medical monitoring program conducted by the Ontario Ministry of Labour will be alluded to in Part B of this chapter. Other data sources regarding the incidence and severity of pneumoconioses will be discussed below.

Attempts by the Task Force on Occupational Lung Disease (Pneumoconiosis)[45]: In order to estimate the population at risk and the incidence as well as the prevalence of pneumoconiosis in Canada, a questionnaire was mailed by the Task Force to all provincial Workers' Compensation Boards. Statistics Canada documents were examined to estimate the population at risk and to assess hospital morbidity as well as mortality due to pneumoconiosis, and special reports were examined.

The data gathered by Statistics Canada during the 1971 census were considered by the Task Force to be the most reliable source for estimating the population at risk (see Appendix 3), however, it was noted that some well-known groups at risk were not included, (e.g. in addition to approximately 14,000 workers employed in mining and milling of asbestos and in the manufacture of asbestos, there were approximately 10,000 shipyard and 4,000 to 6,000 insulation workers directly exposed to asbestos). Since there is no uniform registration of workers exposed to asbestos, silica or other agents capable of causing pneumonicosis, these numbers do not represent the complete picture. In general, they would be a better reflection of the primary (mining, milling) rather than the secondary (processing) industries.

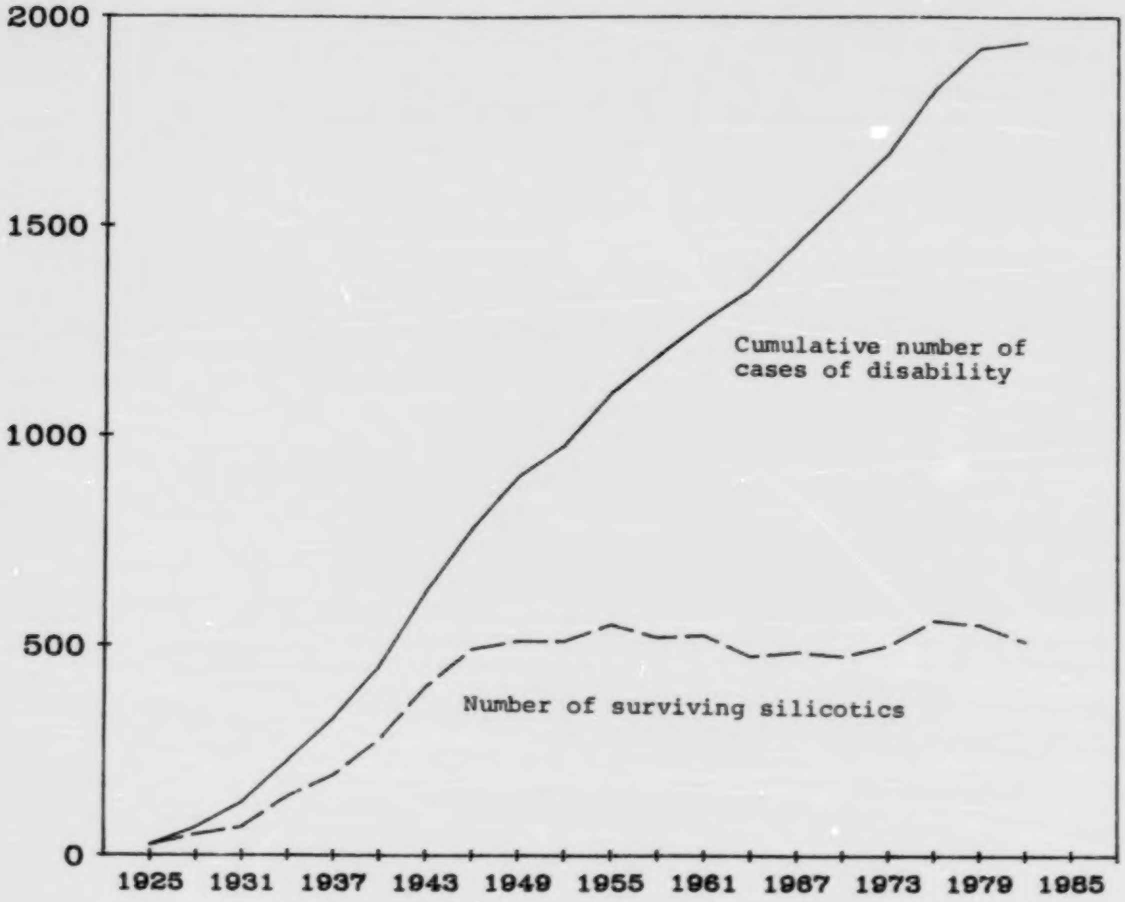
The Task Force examined the number of new cases of pneumoconiosis awarded compensation in each of the provinces between 1965 and 1974 (see Appendix 4 for updated table). The members noted, however, that the year-to-year change in the number of claims, as well as in the number of workers awarded compensation was not a reflection of changes in incidence and in prevalence, but of other factors such as the reporting and adjudication of claims.

The Task Force concluded that it is in fact impossible to estimate the incidence and the prevalence of pneumoconiosis or the size of the population at risk. The reasons for this were discussed in its report; it was pointed out for example that the rapid turnover of the labour force, especially in the younger age groups, results in many workers who were exposed to dust for a few years being lost to follow-up.

In reviewing the mortality data due to pneumoconioses, silicosis was found to be the most frequently reported "cause of death" of the pneumoconioses. Deaths between 1965 and 1974 due to pneumoconiosis in each of the provinces are presented in Table B of Appendix 4, with a total of 307 being reported in Ontario during this period. The most recent figures for each province have also been added. A total of 22 deaths due to pneumoconises were reported in Ontario in 1979.[46] Again, however, the Task Force stressed that mortality statistics probably underestimate the incidence and the prevalence of pneumoconiosis in the population since simple pneumoconiosis does not cause death. When complicated, for instance by cor pulmonale (right heart failure), the existence of pneumoconiosis at the time of death may not be known, or may not be considered relevant and consequently is not recorded on the death certificate. The methodological difficulties encountered by the Task Force attest to the urgent need for better data collection concerning occupational exposures and diseases.

Statistics on Silicosis Among Ontario Miners: Silicosis, caused by the inhalation over a prolonged period of dust containing free silica, has been by far the most serious form of pneumoconiosis among workers in Ontario mines. In 1928, legislative provision was made under The Mining Act of Ontario requiring every person employed in a dust exposure occupation in a mine to have a pre-employment physical examination including a chest x-ray by a medical examiner appointed for the purpose. Surface workers, as well as underground miners are now normally examined annually. The examinations are conducted at miners' chest examination stations located throughout the province. From 1929 to 1974 all stations were operated by the Workers' Compensation Board; on

Fig. IIIA-5


2000
1500
1000
500
0
1925
1931
1937
1943
1949
1955
1961
1967
1973
1979
1985
Cumulative number of cases of disability
Number of surviving silicotics

July 1, 1974, these stations were transferred to the Ministry of Health (Environmental Health Branch) as recommended by the Task Force on Workmen's Compensation in August, 1973.

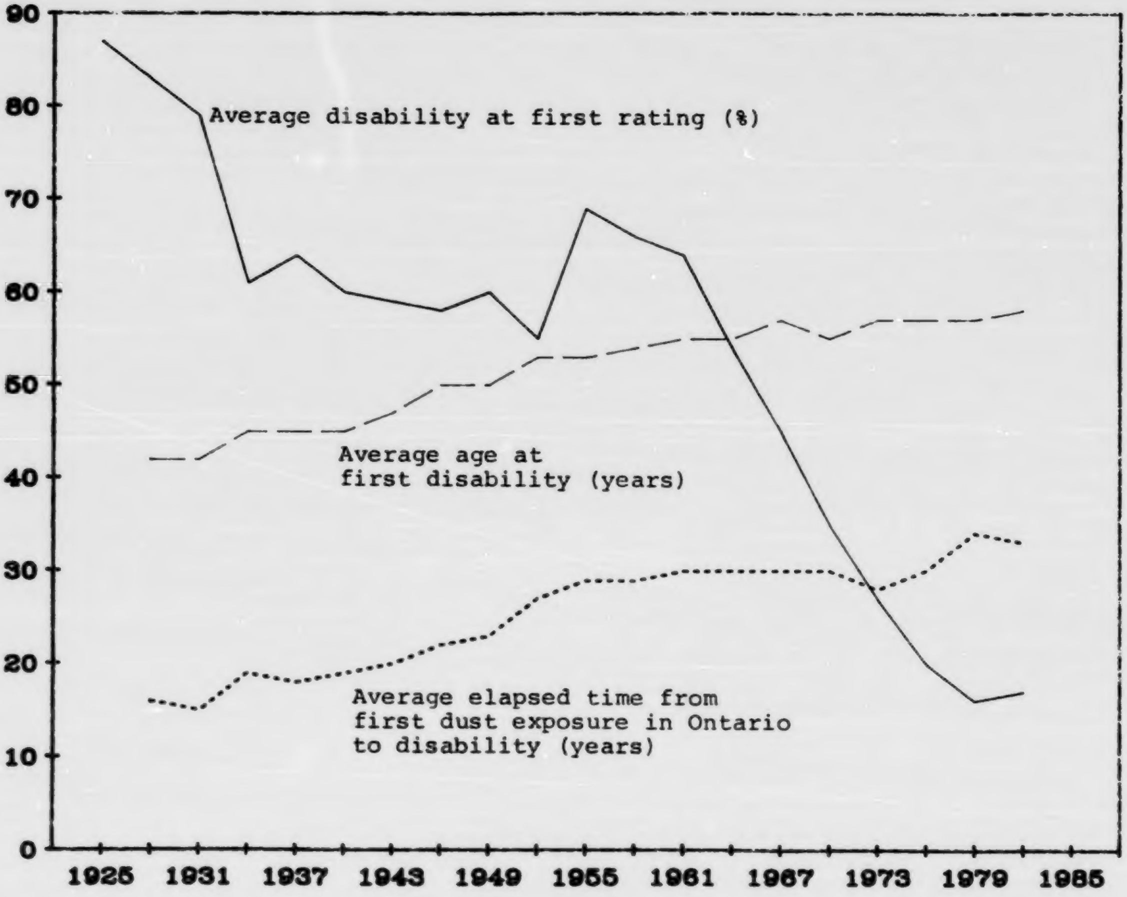
In 1949 a Committee appointed by the Premier of Ontario recommended that a statistical study be made of the reports kept on file at the Workers' Compensation Board. A continuing Committee was consequently set up to prepare and analyze data on the incidence, prevalence and progression of silicosis in the mining industry in Ontario from the reports of the local medical examiners and to study the relationship between age, length of exposure and other factors. In 1950 a Medical Statistics Unit was established at the Workers' Compensation Board and a summary record was devised to handle the individual examination reports for all men with five years or more exposure and for men with x-ray categories of 4 or higher or tuberculosis ratings 13 to 16. (Unlike the International Labour Organization system, a six-point system is used to classify chest x-rays in Ontario; Code No. 4 is "dust effects" or "generalized arborization" and Code No. 5 is "generalized arborization with partial mottling" considered to be the first definite radiological sign of silicosis. The frequency of new 5 ratings is used to provide data on the incidence of radiological silicosis and is not related to disability from silicosis or to claims established, as will be discussed later in this chapter.)

The mining statistics program, computerized from 1968, was last reported to contain information on over 700,000 examinations for some 80,000 men.[47] Data on the incidence and prevalence of radiological silicosis for each type of metal mined in Ontario (gold, nickel-copper, silver, iron and uranium), and for each combination of metals, can be analyzed. Two reports have been prepared so far, one confined largely to the presentation of data for Ontario gold mines (September 1976)[48] and a similar report prepared for men exposed to dust in Ontario uranium mines (March 1976).[49] The latter report was presented to the Advisory Committee on Occupational Health of the Ontario Mining Association in October, 1976, and has since been updated to include statistics to the end of 1977.[50] These statistics were also used in the studies prepared for the Ham Commission on Health and Safety in the Mines. More recent figures are presently unavailable as they are being used in yet a further study in this area.

It should be noted that silicosis and other related pneumoconioses are not confined to mining. For example, from 1972 to 1974 inclusive, mining accounted for only slightly more than half the cases of pneumoconiosis recognized by the Workers' Compensation Board.

Almost 2,000 cases of silicosis (5 ratings) among miners have been acknowledged in Ontario since the program began. The numbers increased from 1930 to 1934 through 1940 to 1944, declined slightly in 1945 to 1949, sharply in 1950 to 1954, and had remained at or below the 1950 to 1954 level since that time, until they increased again in the period beginning in 1965. This

Fig. IIIA-6



90
80
70
60
50
40
30
20
10
0
1925
1931
1937
1943
1949
1955
1961
1967
1973
1979
1985
Average disability at first rating (%)
Average age at first disability (years)
Average elapsed time from first dust exposure in Ontario to disability (years)

Table IIIA-1

Mortality Among Miners Receiving Workers' Compensation for Silicosis

(1940 - 1975)[a]

Cohort[b]	All Causes[c] Observed	All Causes[c] Expected	SMR	Non-Malignant Respiratory Diseases (Excluding TB) Observed	Non-Malignant Respiratory Diseases (Excluding TB) Expected	SMR
1940 - 1949	383	175	219	112	10.3	1,091
1950 - 1959	224	145	154	66	10.6	623
1960 - 1969	119	95	125	43	7.9	544
1970 - 1975	31	185	168	10	1.4	714
Total	**757**	**433.5**	**174**	**231**	**30.2**	**765**

a - Source: Finkelstein, M. Kusiak R. and Suranyi, G. Mortality among miners receiving workmen's commpensation for silicosis in Ontario: 1940-1975. J.O.M. 24:663, 1982.

b - Cohorts of compensated silicotics based on the decade of initial disability rating. Each worker was followed-up until death or the end of 1978. The authors chose not to stratify the cohorts according to a "percentage disability" rating "because of the subjectivity of this assessment, the possibility of variability of this assessment over the 36-year entry period to the study and its nontransposability to other jurisdictions".

c - A more detailed analysis of cause of death is provided by the authors.

Table IIIA-2

Mortality Among Asbestotics

	Asbestos-Cement Workers						All Certified Asbestotics		
	251 Man-Years Observation[a]			388 Man-Years Observation[b]			733 Man-Years Asbestotics[c]		
Cause of Death	Observed	Expected	O/E	Observed	Expected	O/E	Observed	Expected	O/E
All Causes	24	5.1	4.7	29	6.9	4.2	61	16.6	3.7
All Malignancies	12	1.3	9.2	12(14)[d]	1.7	7.1	22(24)	4.0	5.5
Lung Cancer				6(4)	0.6	10.0	11(9)	1.4	7.9
Pleural Mesothelioma	7	0.5	14.0						
Peritoneal Mesothelioma	2			2(7)	1		3	1	
Gastrointestinal Cancer				1(1)	0.4	2.5	2(2)	1.0	2.0
Non-Malignant Respiratory Disease	5	0.3	6.0	7(6)	0.4	17.5	23(22)	1.1	20.9
Ischemic Heart Disease	3	2.0	1.5	4(4)	2.8	1.4	8(8)	6.7	1.9

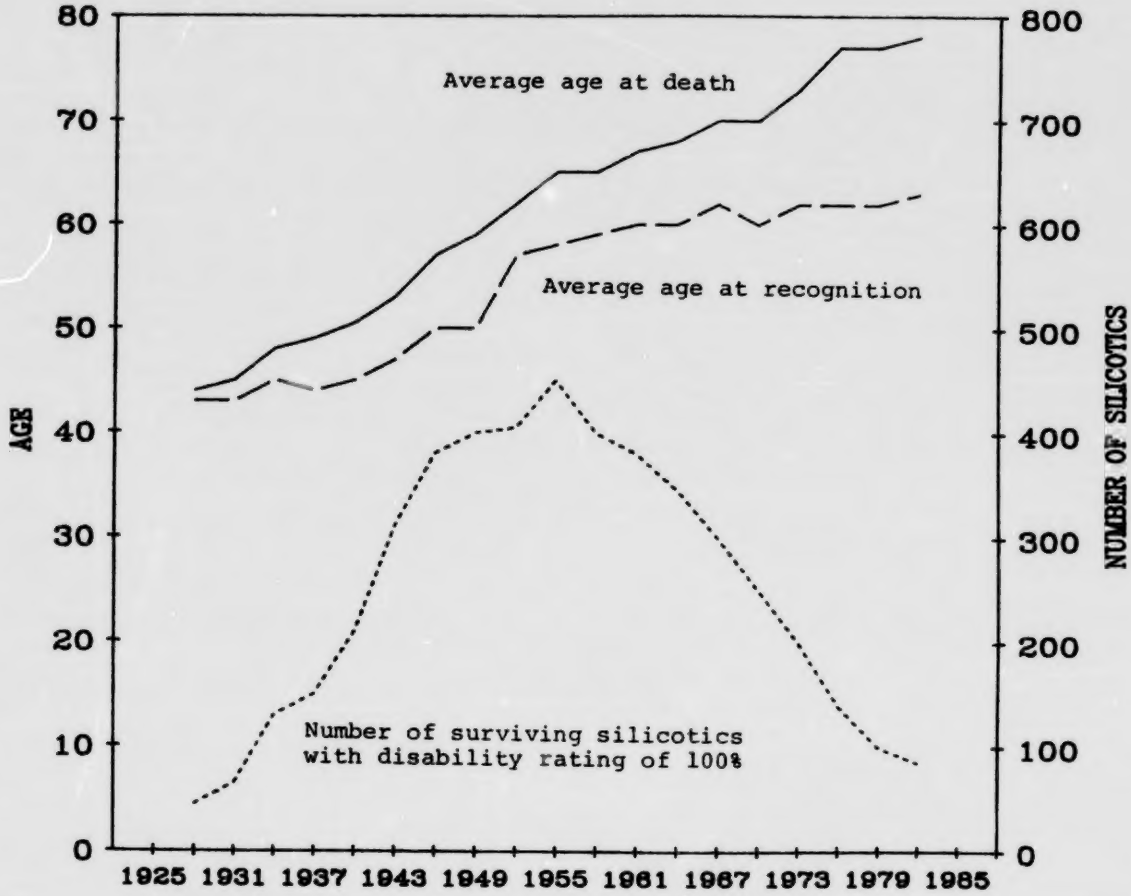
a Source: Finkelstein, M. Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory. Ont. Ministry of Labour, unpublished. (The cohort consisted of those employed prior to 1960, exposed for a minimum 15 years. Follow-up ended July 1980. N=61 asbestotics.)

b. Source: Finkelstein, M., Kusiak, R. and Suranyi, G. Mortality Among Workers Receiving Compensation for Asbestosis in Ontario. C.M.A.J. 125: 259, 1981. (The cohort consisted of all asbestotics compensated prior to July 1979 who had been employed as asbestos-cement workers. N=91.)

c Source: Finkelstein et al, 1981, op. cit. (The cohort consisted of all asbestotics compensated prior to July 1979. N=172.)

d Numbers in parenthesis represent number of deaths when cause was changed because of additional evidence available to the Compensation Board.

Fig. IIIA-7


Average age at death
Average age at recognition
Number of surviving silicotics with disability rating of 100%
AGE
NUMBER OF SILICOTICS
80
70
60
50
40
30
20
10
0
800
700
600
500
400
300
200
100
0
1925
1931
1937
1943
1949
1955
1961
1967
1973
1979
1985

increase is due to uranium mining which commenced in Ontario during the mid-1950s (see Appendix 5, Table A)[51].

At the urgent request of union locals of the United Steelworkers of America, the Ministry of Health conducted a study on the state of the respiratory organs of mine workers in Elliot Lake. This showed that among about 1,000 dust exposed workers employed in the study period, 36 or 3.6% of the total had a radiological classification of silicosis and an additional 53 or 5.3% had an x-ray code 4.[52]

It has been noted by Workers' Compensation Board personnel however, that of 255 Ontario uranium miners with new 5 ratings discovered to the end of 1977, all but 88 had additional exposure outside Ontario and of these 88, all but 14 had exposure to other Ontario metals (see Appendix 5, Table A).[53] Similar results were found for the 428 new 4 ratings in this group of miners (see Appendix 5,Table B). Despite the fact that heavy exposure takes place outside Ontario and in mines outside Elliot Lake, and despite the fact that miners, being a fairly mobile group are subject to these exposures, it can nonetheless be concluded that silicosis from the dust conditions in Elliot Lake uranium mines is still a serious problem. It was also specifically pointed out that the vast majority of the miners who developed silicosis had dust exposure in Ontario gold mines. In fact, 90% or 1,501 of the new 5 ratings and 3,225 (85%) of the 3,811 new 4 ratings discovered by 1974 had exposure in Ontario gold mines (see Appendix 5, Tables C and D)[54] indicating a problem in this area as well.

An updated version of the graph developed for the Ham Report illustrating the cumulative number of new cases of silicosis and cumulative number of surviving silicotics since 1925 is presented below at Figure IIIA-5. It should be noted that while there were 1,876 workers with radiological silicosis, only 1,848 have so far received compensation awards. (The other 28 are not included as "cases" of silicosis.) Due to the long average latency and the relative continuity of historical dust conditions it can be concluded from these data that a significant number of cases of silicosis will continue to appear (see Figure IIIA-5).

The number of new cases recognized annually has been shown to be approximately equal to the number of silicotics dying per year, with the population of surviving silicotics averaging 570 over the 25-year period from 1950 to 1974. Approximately 49% of the total mine employment figure consisted of those employed in a "dust exposure occupation". The numbers have fluctuated over a time, but averaged 17,500 for the period 1950 to 1974. The growth prevalence factor for silicosis was calculated as 3.3% of the dust exposed population as a whole[55], consistent with previous calculations.

The impact of silicosis on the lifespan and well-being of workers who develop the disease varies considerably, depending on age, severity and co-existing health problems. The average age at which disability was first recognized has increased from age 43 in 1946 to age 56 in 1974, possibly indicating that disability is not developing as early in the life of silicotics. The number of surviving silicotics on 100% permanent disability benefits has also decreased (see Figure IIIA-6), but these figures are clearly subject to trends in adjudication. Moreover, not only is silicosis still very much present, but disease other than silicosis is often still present in silicotics. It has been noted, for example, that tuberculosis was present in 5.5% of silicotics who died in the period 1970 to 1974, a rate much higher than the comparable figure for the general population.[56] Furthermore, as shown in Table IIIA-2, Dr. Murray Finkelstein of the Special Studies and Services Branch of the Ontario Ministry of Labour, with the aid of Program Planning and Statistical Service Branch of the Ontario Workers' Compensation Board has found that while there were about 434 expected deaths among the 1,190 Ontario silicotics who were compensated between 1940 and 1975, 757 deaths were actually observed (SMR of 174). Moreover, while only about 30 deaths from nonmalignant respiratory disease (excluding TB) were expected, 231 were observed (SMR of 765), thus attesting to the serious nature of the disease and/or concomitant respiratory conditions.[51]

Based on the average increase in elapsed time from first dust exposure in Ontario to the diagnosis of silicosis, the increased age at the time of recognition and a decreased average percent initial disability awarded in compensation (see Figure IIIA-7), the Ham Commission concluded that the average severity of silicosis following its recognition is declining. Policy in the adjudication of compensation claims certainly plays a role in this statistic as well and will be discussed more fully in Part B of this chapter. Suffice it to say that despite some encouraging trends, silicosis still remains a significant problem in Ontario mines, and hence, still requires careful consideration in the compensation system. (The extent of silicosis in Ontario foundries and other secondary industries has not been studied.)

Studies of Ontario Asbestos Workers: No serious estimate of the number of workers exposed to asbestos in Ontario has yet been published however, some studies relating to the seriousness of asbestos exposure in particular Ontario workplaces are noteworthy. It has often been stated that asbestotics have a decreased life expectancy and suffer more from other conditions than those who have had no significant asbestos exposure. This has been borne out by the work of Finkelstein and his colleagues. First, the mortality of 172 workers who had received compensation for asbestosis in Ontario was investigated.[58] The mean age at the time of award was about 56 years and, unlike the findings for silicotics, there was no age difference between those who had received compensation in earlier years compared to those more recently compensated. It was found that these asbestotics had an increased rate of death relative to the general population for non-malignant respiratory diseases as well as malignancies, as shown in the last 3 columns of Table IIIA-2, with non-malignant respiratory diseases the leading cause of death. (The authors made the significant observation that malignant mesothelioma was under-represented as a cause of death in the official coding.)

Survival for asbestotics from the date compensation was awarded was only 69% of that expected at five years and 53% at ten years, thus confirming that certified asbestotics indeed have a decreased life expectancy. The increase in mortality rates ranged from 3-fold among men of retirement age to 19-fold among men aged 35 to 44.

A comparison of mortality among the 91 certified asbestotics who had been production workers in an Ontario asbestos-cement factory, with that of the Ontario population again showed markedly elevated mortality rates, despite the fact that these workers tended to be older at the time of their compensation award compared to insulation workers who constitute the other major group of workers who developed asbestosis. As shown in the middle portion of Table IIIA-2, respiratory disease and malignancies were also primarily responsible for the increased mortality rate.

Secondly, Finkelstein also had studied the probability of development of compensable asbestosis among the 201 workers at an Ontario asbestos-cement factory, first exposed to asbestos prior to 1960 and employed a minimum of 15 years.[59] By July 1980, 39% of the production workers (61) and 20% of the maintenance workers (9) had developed a compensable chest disability. The "latent interval" generally exceeded 20 years, however one heavily exposed worker was certified after 13 years and died four years later of cor pulmonale. Finkelstein calculated the cumulative probability of developing compensable asbestosis by the end of the 32-year follow-up period, and found that it was indeed related to the length of time spent in dust exposure. Specifically, probability was 68% for men exposed 15 or more years, 37% for an exposure of 11 to 14 years, 13% for those exposed 6 to 10 years and 28% for those exposed 5 or fewer years. (The small number of men in the shorter exposure categories accounts for the considerable statistical fluctuation.) Finkelstein admits that the extrapolations used in exposure assessment are crude and probably overestimate the risk of asbestosis at low exposures, but he states that this would be balanced by the fact that the end point was "certified disability, an advanced stage of asbestosis." He also pointed out that the major risk at lower exposure is from cancer rather than asbestosis.

A comparison of mortality among the 61 certified asbestotics with that of the Ontario population again showed markedly elevated mortality rates, with deaths due to respiratory disease and malignancies being again primarily responsible (see first section of Table IIIA-2).

Thirdly, a mortality study of a group of 339 workers of an Ontario asbestos-cement factory hired prior to 1960 and who worked for a minimum of nine years was also undertaken. A group of 87 who worked for the company, but were not exposed to asbestos had mortality rates similar to that of the general Ontario population, while the group of asbestos-exposed employees had an all-cause mortality rate double that of the Ontario population. Mortality rates due to malignancies were five times higher, and deaths attributed to lung cancer were eight times the expected rate. According to the model developed, "men exposed to dust at a concentration of 1 fibre per cc for twenty years beginning at age 25 might experience, between the ages of 45 to 60, a risk of death from mesothelioma and lung cancer increased 30% to 250% above the risk faced by unexposed men."[60] It should be noted, however, that the reliability of this estimate is very weak due to inadequate exposure data.

A better understanding of the scope of the asbestos problem in Ontario will no doubt be forthcoming with the report of the Dupre Commission on Asbestos.[61]

3.2 Chronic Obstructive Lung Diseases

3.2.1 *The Pathogenesis, Clinical Presentation and Natural History*

There is much confusion and debate within the medical profession over the terminology and diagnostic criteria defining each of the diseases in the chronic obstructive lung disease group. It is not uncommon for someone to be told by one doctor that he or she is suffering from chronic bronchitis, by another that the disease is emphysema and by a third that the problem is bronchial asthma. Often characteristics of all three of these diseases may be present. The lack of agreement regarding definitions of each of these clinical entities has led to much confusion regarding the cause of these diseases (etiology), the damage they produce, (pathophysiology), their prognosis and their prevalence.

The chronic obstructive lung diseases consist primarily of chronic bronchitis and emphysema. Chronic bronchitis refers to the condition of chronic excessive mucous secretion in the bronchial tree. The word 'chronic' was defined as production of sputum on most days for at least three months in the year, during at least two years.[62] Irreversible, its onset is gradual, not usually causing disability before age 40. (Some have pointed out that people with chronic cough may never in fact have significant airway obstruction. Although a chronic cough is certainly bothersome, it is the obstructed airways that cause serious breathing difficulties.) Emphysema, often associated with chronic bronchitis, is characterized by the enlargement of the air spaces beyond the small bronchioles with destruction of the alveoli (air sacs). It is inevitably disabling. Asthma, also an obstructive lung disease, is characterized by the reversible narrowing of air passages, which may or may not lead to permanent damage.

Chronic Bronchitis: A person who develops chronic bronchitis often first notices cough and sputum production in the mornings, shortness of breath on exertion and occasional wheeze. Often chronic inflammatory changes develop first in the smaller airways. Later, the mucous glands in the large bronchi become increasingly thickened, and the resistance to airflow becomes more severe. The patient may note that his or her cough is getting worse with the sputum becoming more copious, and that it has become increasingly difficult to breath.

Variations in airway resistance in different regions of the lungs leads to some areas being underventilated and other areas overventilated in relation to their blood flow. In the areas which are underventilated, the amount of oxygen in the blood falls. This may cause the blood vessels in this area of the lung to constrict. At first this vasoconstriction is reversible; later pulmonary hypertension results. This increased resistance in the blood vessels of the lungs in turn causes an increased strain on the right side of the heart (which has to pump blood to the lungs) and right heart failure (cor pulmonale) may result. The strained right heart in turn places greater strain on the left side of the heart, (responsible for pumping blood to the rest of the body) and the patient may eventually suffer from cardiac failure. In those people whose pulmonary blood vessels are less reactive, cor pulmonale may not develop, but their oxygen concentration may fall to dangerously low levels and lead directly to respiratory failure. At this stage the patient may require hospitalization and oxygen therapy.

Meanwhile, in the areas of the lung which are overventilated in relation to blood flow, the ventilation of what is called the physiological dead space increases the work of breathing and causes the patient to experience even greater shortness of breath.

The natural history of chronic bronchitis is characterized by a general pattern of steady deterioration which is extremely variable. The patient may experience increasingly frequent and severe respiratory infections, increasing shortness of breath and incapacity, and finally edema and cor pulmonale. By this point the patient may have swollen legs and abdomen, and may even have a drowning feeling from fluid build up. Respiratory infection may also trigger episodes of respiratory failure, in which the oxygen concentration falls and carbon dioxide is retained. The patient's family and friends may even note that the patient has cyanosis, a bluish colour depicting oxygen lack.

Some investigators have used the volume of sputum expectorated in the first hour upon arising from bed as a rough estimate of the severity of chronic bronchitis.[63] Other indicators of severity of the disease are the number of chest infections and the purulence of the sputum.[64] None of these measurements, however, hold a high degree of accuracy, and the x-ray at this stage may also be entirely non-contributory.

In pathological specimens, the diagnosis of severity of chronic bronchitis is measured with the use of indices such as the Reid Index utilizing the ratio of thickness of the mucous-secreting glands to the wall thickness, since in bronchitis the former becomes hypertrophied (i.e. enlarged) merely as a result of irritation. Infection may, however, supervene and when it occurs in chronic bronchitis it may result in permanent damage to the airways and surrounding tissue. Chronic bronchitis may also lead to the destructive changes of emphysema.

The natural history and prognosis of chronic bronchitis is thus still quite unclear and it is largely related to the prevalence of infection, the development of emphysema, or the progression to a more severe form of chronic bronchitis with ventilaton-perfusion disturbances and cor pulmonale. It is not known what proportion of chronic bronchitics progress to this stage.

While it is well recognized that the cough and sputum of cigarette smokers' bronchitis will clear up after a few months after the person stops smoking, this is not true of the physiological abnormalities associated with concomitant emphysema, and although there is the clinical impression that removal of a worker with bronchitic symptoms from exposure to dust is associated with a decline in the severity of these symptoms, this is difficult to document.[65] The established reduction in ventilatory capacity induced by industrial bronchitis, is not reversible but removal from exposure may slow the progression.

Emphysema: This disease is characterized by distension of the air spaces distal to the terminal bronchioles with destruction of their walls. There are two main categories of emphysema: panlobular where the destruction involves the whole periphery of the lobule and all regions of the lung can be affected; and centrilobular, where distension is limited to the respiratory bronchioles and alveolar ducts, tends to be more severe at the lung apices and is very common, at least in mild form, especially in the presence of bronchitis. It is unclear whether the two types produce significantly different clinical features. (In other words, to the patient the disease is essentially the same in either case.)

The patient generally presents with a history of effort intolerance due to shortness of breath; cough may be absent or productive of sputum depending on the degree of concomitant bronchitis. Signs of chronic airway obstruction are found as discussed above. Physical examination may also reveal a hyper-resonant, barrel-shaped chest with poorly heard breath sounds, but these are unreliable in establishing the degree of emphysema. A chest x-ray may also show the signs of hyperinflation (low flattened diaphragms with a long narrow heart shadow and increased retrosternal translucency) and the attenuation and narrowing of the peripheral pulmonary vessels correlate to some extent with the severity of emphysema. Signs of heart failure may be present to complicate the picture (i.e. enlarged heart, pulmonary congestion, etc.). Pulmonary function tests show airway obstruction, increased residual volume and increased total lung capacity, as well as reduced diffusing capacity. Oxygen desaturation during exercise often occurs. (In contrast, in pure bronchitics, the TLC is frequently normal, the diffusion capacity is not severely impaired and although there is often a low resting oxygen pressure, it may rise as the ventilation-perfusion relationship improves during exercise). As is true for chronic bronchitis, the prognosis of a patient with emphysema is dependent on the prevalence of infection and the development of cardiac complications.

Emphysema is a difficult diagnosis to make during life and it is often difficult to determine how much of the defect in function is attributable to emphysema and how much to bronchitis. In fact, there is even a large intra- and inter-observer variation in the pathological assessment of emphysema. Its importance, however, is clear, as this disease is steadily increasing, it is not reversible and there is little that can be done by way of treatment once it is manifest.

Emphysema can develop as a complication of chronic bronchitis or any other lung condition in which the airways become seriously obstructed. Cigarette smoking, air pollution, infection and occupational factors have all been implicated etiologically. The fact that a certain percentage of the population has an inherited enzyme deficiency that predisposes them to emphysema has captured the attention of the Ontario Workers' Compensation Board. This group of people have been singled out in compensation guidelines and therefore the knowledge surrounding the deficiency of this enzyme will be reviewed in some detail in Part B of this chapter.

Asthma: The phenonomen of hypersensitivity or atopy and the mechanisms that cause allergic reactions of the lung were discussed in Section 2.4. Essentially, asthma is characterized by airway obstruction, reversible spontaneously or by treatment and is due to an increased reactivity of the airways to a variety of specific or non-specific stimuli. These stimuli may be of occupational origin (isocyanates, enzyme detergents, red cedar dust, hemp dust, organic gums, soldering fluxes, etc.) or season variances (such as pollens, molds, and fungi) or household factors (such as pets or dust). Symptoms may also be induced by exercise, exposure to cigarette smoke or dusty atmospheres.

Bates preferred to divide asthmas into two types: "spasmotic asthma" in reference to sensitivity to inhaled allergens resulting in wheezing, and "chronic infective asthma" in reference to an underlying condition of chronic bronchitis with attacks of severe bronchial spasms usually induced by respiratory infection.[66] Some prefer to use the term "asthmatic bronchitis" for the latter. Bates states that spasmotic asthma usually begins in childhood or early adulthood as a result of an inherited atopic predisposition. He believes that "when asthma begins after the age of 40, it should be considered as due to factors other than allergy until proven otherwise," i.e. is "intrinsic". The sensitization to occupational exposures however, does not always occur in atopic individuals and, in fact, for some potent sensitizers such as isocyanates, the condition of atopy shows little correlation with the development of symptoms[67]. In this situation the condition may, indeed, be of an allergic nature, but develops in adulthood nonetheless due to the pattern of exposure.

The cardinal symptom of occupational asthma is a complaint of wheezing at work. The worker notes chest tightness, shortness of breath and often a wheeze can be heard by others around the worker. However, often the symptoms do not develop until hours after exposure, in the evening or even in the night. The clinical patterns of occupational asthma are varied. It typically occurs in an individual who has worked in the industry a number of years and who may have had some previous upper respiratory symptoms such as rhinitis (stuffy nose). In the classic case, the worker tends to improve on weekends and vacations and is worse as the workday and week progresses. In addition to shortness of breath and rhinitis, cough and sputum often occur.

If the substance in the workplace is a known allergen, the clinical history should be adequate to establish a diagnosis. However this is often not the case. As Seaton points out: "Whenever a patient blames dust or conditions at his workplace for his respiratory illness, he should be taken seriously and appropriate investigations should be undertaken. Only in this way have most of the causes of occupational asthma been described and in many instances, has the hazard to health been reduced or eliminated."[68]

Examining the patient between attacks is generally unhelpful and lung function tests also are often normal, though there may be some residual airway obstruction. It is useful to examine the patient during exposure if at all possible or to measure the peak flow rates during the attack as compared to when symptom-free. Measurements pre- and post-shift may be helpful. Some people believe that the diagnosis of occupational asthma could not be made reliably on clinical history and requires a "provocation test".[69] This consists of administering to the worker a preparation of the suspected allergen to see if it produces the symptoms or results in bronchial spasm. There are dangers and difficulties involved in these procedures and they are by no means universally accepted for routine use. Some believe that it is safer to simply remove the worker from exposure for a trial period and then observe changes upon reintroduction into the workplace. This approach has proven problematic from the compensation point of view, as will be discussed in Part B of this chapter.

When it is confirmed that a worker has developed asthma in response to an occupational exposure, he or she should be removed from exposure to the allergen. (Needless to say, the amount of potential allergen in the workplace should be reduced to a minimum by effective ventilation and enclosure of the processes whenever possible.) Although the bronchospasm is reversible by definition, the occupational asthma may lead to residual damage. Little data exists as to how frequently this occurs. Moreover, once bronchial hypersensitivity has developed to one substance, the airways tend to react to other non-specific irritants and bronchial spasm may even be triggered on exertion in some individuals long after removal from the initial sensitizer.[70] This too has important implications for compensation, as although no clear "disability" is manifest, the patient is disabled by having to avoid many activities or situations as a result of this condition.

In summary, some people are more likely to develop asthma than others. Nevertheless, some workers who develop asthma in relation to their occupation have no particular predisposition and with some occupational allergens, no correlation with "predisposition" has ever been shown. In other situations, workplace factors may "non-specifically" aggravate a person's asthmatic condition. Moreover, although residual airway impairment may or may not result, the individual's bronchial tree is permanently altered once sensitization has occurred. This has implications regarding future job prospects well beyond the need to avoid further exposure to the specific sensitizer. Issues with respect to establishing the occupational origin of the symptoms are particularly controversial and relate to a problem area in the Ontario workers' compensation system. This will be discussed in Part B of this chapter.

3.2.2 *Prevalence of Work-Related Chronic Obstructive Lung Diseases*

Cigarette smoke, air pollution and various workplace dusts, fumes, and gases (e.g. welding fumes, metal dusts, sulphur dioxide, etc.) have all been associated with obstructive lung diseases. Some leading experts infer that of all the various respiratory diseases brought about by exposure to airborne particulates in industry, chronic bronchitis is probably the most common. In an individual case it is rarely possible to even approximate the relative contribution of each of the many responsible factors, however, there is reason to believe that workplace factors play a larger role in the sum total of morbidity from these diseases than is presently recognized by compensation figures.

The evidence that suggests this comes from a variety of sources. First, there are experimental studies linking specific hazards known to occur in the workplace with damage to the respiratory tract. For example, studies have shown the deleterious effect of sulfur dioxide, ozone, chlorine and many other substances on the bronchial airways. As noted in Chapter I, Part A, the number of chemicals, physical conditions and processes which have been suggested as hazardous to the respiratory system probably is in the hundreds, if not thousands.

A second source of evidence alluding to the role of industry in chronic bronchitis is the inter-regional difference found between cities in which heavy industrial pollution occurs and those in which this exposure is absent. One such study, to be described below, showed that the incidence of chronic bronchitis in Sudbury significantly exceeds that in Ottawa.

Thirdly, there are an increasing number of studies that are revealing inter-occupational differences in the incidence of chronic obstructive lung disease. One Ontario study, to be described below, showed that workers in one particular nickel converter plant had a higher incidence of chronic obstructive lung disease than those elsewhere. Another study of Elliot Lake miners suggested that an increased incidence may be associated with this occupation as well. As will be discussed below, at least a dozen studies have documented an increased incidence of chronic obstructive lung disease in coal miners, and an increased incidence has also been found in foundry workers, welders, asbestos miners, gold miners, coke oven workers, hemp workers, rubber-industry workers, chemical-industry workers and workers exposed to cotton dust. In fact, it is generally accepted that any heavy dust exposure leads to increased morbidity from chronic bronchitis.

A fourth source of evidence suggesting the under-recognition of occupational respiratory disease comes from surveys such as that performed by Discher et al[72] for NIOSH, outlined in Appendix 2 of Chapter I, Part A. A specially-trained medical team found that 12.2% of the workers exposed to known respiratory irritants had respiratory impairment; this rose to 16.5% when they considered only those whose exposure was not adequately controlled. Overall, they found that 16.9% of all the respiratory disease found among the population they surveyed was probably of occupational origin, (despite the fact that only 1 worker of the 49 identified as having an occupational respiratory disease filed a compensation claim).

Yet a fifth source suggesting that workplace factors in respiratory disease have been understated comes from the Survey of Disabled and Non-disabled Adults[73], which was also discussed in Chapter I, Part A. Based on a self-assessment of the work-relatedness of their disease, it was found that 16.1% of individuals disabled with respiratory disease felt that their condition was work-related, despite the lack of financial incentive to do so. Although the weakness of the methodology is apparent, tests for internal and external consistency led the authors to the conclusion that these results had validity. For example, 78% of those with respiratory disease who reported work-relatedness, were found to have been in occupations with heavy exposures to dusts and fumes.

A survey in Britain[74] resulted in similar conclusions. For every case of occupational disease which was compensated, there were at least 5 episodes of illness that workers attributed directly to their workplace for which compensation was never received. Most of these were respiratory, diagnosed as chronic bronchitis.

In Great Britain, chronic bronchitis has been recognized for many decades as a major cause of incapacity for work and much British data has been accumulated. One estimate states that 8-17% of the male population and 3-8% of the female population suffer from chronic bronchitis.[75] In the United States and Canada, the disease has been given much less attention although the crude death rate from emphysema with or without chronic bronchitis has risen faster during the last 15 years than any other cause of death including lung cancer. (It should be noted, however, that changes in the classification of diseases in this group preclude any real quantification of this trend.) Dr. A. H. Sellers of Ontario has noted that the greatest increase in emphysema has occurred in men over the age of 55 years. Among men aged 55 to 64 he states that the death rate tripled; it was four-fold for those 65 to 75 and more than eight fold for those over 75 years old.[76] Almost certainly the magnitude of this increase is artificially inflated by changes in diagnostic criteria, however, with an increasing incidence of emphysema in the older male age group, an increasing number of workers in dusty trades will also develop the condition. The question this poses is whether the presently occurring increase in chronic bronchitis and emphysema can be documented to be greater among those exposed to dust and other industrial contaminants, than among those not exposed.

The Sudbury/Ottawa Study[77]: From 1969 to 1971, a household survey for chronic obstructive lung disease was carried out in Ottawa as the first of a series to cover a number of Ontario localities between which there was a fair difference in the amounts or type of air pollution. The second city selected for the studies from 1972 to 1973 was Sudbury, housing the world's largest nickel and copper smelting operation, which despite recent improvements was then emitting more than twice as much sulphur dioxide daily as was New York City.

Lung function tests of 3,288 Ottawa residents and 2,208 Sudbury residents were performed. These consisted of measuring the forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1), and calculating the ratio of FEV_1/FVC as an assessment of airway obstruction. The results showed that the mean FEV_1/FVC was appreciably lower in Sudbury, as shown in Appendix 6. This difference was statistically significant for both men and women considered separately and held true when age and smoking habits were taken into account. The prevalence of symptomatic chronic bronchitis was significantly higher in Sudbury males than in Ottawa males, but no such difference was detected in females (See Appendix 6). Differences in ethnic origin, climate, or dates in which the study was performed were considered by the authors as unable to account for the differences in the result, which they felt were predominently due to air pollution exposure. In a similar study comparing Chilliwack, B.C. (taken as free of air pollution) to Berlin, New Hampshire (which has significant sulphur dioxide and particulate exposure)[78] lung function was found to be significantly worse in the residents of the more polluted city, however prevalence of symptomatic disease did not differ. The question was therefore raised as to whether the higher rate of symptomatic disease in Sudbury males may at least be partly attributable to occupational factors rather than to the general level of air pollution alone.

Inter-Occupational Differences: Several studies have been carried out in Ontario in an attempt to clarify the relative contribution of various occupational exposures. Some of these were directly prompted by concerns articulated in labour union briefs and militant wildcat strikes in northern Ontario, and were highlighted in the Ham Commission on health and safety in mines.[79]

Chronic Obstructive Lung Disease Among Workers Exposed to Sulphur Dioxide in one Nickel Converter Plant: In 1970 a study of morbidity and mortality among workers at a Copper Cliff Nickel Converter Plant was completed by Dr. R. B. Sutherland of the then Ontario Department of Health.[80] An increase in the frequency of absences attributed to diseases of the lower respiratory tract was noted in men with over 20 years exposure in the converter area. These findings suggested that long-term employment in a converter area might bring about an increase in chronic respiratory disease with periodic exacerbations leading to more frequent absences from work. In an attempt to ascertain whether indeed this was so, the study was supplemented in August/September 1973 by a survey of men employed at that time for ten years or more in the converter area, comparing them to a similar group taken from the copper refinery. This study showed that both subjective symptoms and objective signs of chronic lung disease had greater prevalence in the converter group which could not be explained by smoking alone.[81]

Air sampling was conducted which showed that workers in the converter area were significantly exposed to sulphur dioxide gas, metal fumes and dust. Dust was not considered to be present in any significant quantities in the copper refinery, and the concentration of sulphur dioxide was generally below the level of detection of the sampling instruments. The two groups matched well for age, height and length of employment. The participants were divided according to their smoking histories, into those who had "never smoked", "former smokers", and "smokers". The survey consisted of a medical history obtained with the use of a modified British Medical Research Council questionnaire, an occupational history and simple lung function tests.

The results indicated that symptoms of chronic bronchitis among converter workers were 14.8% higher than among refinery workers, this difference being even more pronounced (23.5%) when comparing smokers (see Appendix 7, Table A). All three parameters of lung function (FVC, FEV_1, and FEV_1/FVC_1) were lower in converter than in refinery workers even though the lower average values found in converter workers were well within the accepted "range of normal" values. Again, the differences were more pronounced in the smoking group (see Appendix 7, Table B). Moderate to severe airflow obstruction (FEV_1/FVC at or below 59%) was found only in the converter group, 8.4% of whom had this degree of impairment (see Appendix 7, Table C). Maximum Mid-Expiratory Flow Rate (MMFR) was also performed as a more sensitive indicator of airflow obstruction. Again a mean difference was detected to the disadvantage of the converter workers. It was thus concluded that the increased morbidity due to diseases of the lower respiratory tract found by Dr. Sutherland was indeed attributable to repeated exacerbations of chronic obstructive lung disease and was work-related.

This conclusion was found to be consistent with other studies on the subject, and it was recommended that chronic obstructive lung disease among converter workers exposed to sulphur dioxide be accepted as a compensable disease. Precise guidelines will be discussed in Section B of this chapter.

Chronic Bronchitis in Workers Exposed to Mineral Dust (with or without Diesel Fumes) in Elliot Lake Uranium Mines[82]: In response to a request by the United Steelworkers of America, a survey was conducted by the Ministry of Health in 1974 to determine the effects of the occupational environment on chronic lung disease among underground and surface workers at two uranium mines at Elliot Lake. A total of 1,043 exposed workers were identified, 973 (93%) participated. For the sake of completeness, efforts were made to assess chest x-ray findings of those who did not participate. (These results were given in Section 3.1.2 in the discussion of silicosis). Each participant was examined by means of a questionnaire, lung function tests and chest x-ray.

The overall prevalence of chronic bronchitis was 12.1% (the details of which are provided in Appendix 8, Table A). The incidence of chronic bronchitis was found to be higher among smokers, even those younger than 25 years of age, than among former smokers and those who never smoked. It is, however, also seen to rise with age, which was in turn found to correlate highly with length of exposure. A particular problem was that while the years of exposure were known, the intensity of the dust exposure involved could not be taken into account. A statistical analysis was performed, in an attempt to sort out the effect of the different factors. As shown in Appendix 8 Table B chronic bronchitis was found to be significantly and separately related to both smoking and the mine environment. The table also indicates that breathlessness is associated with occupational exposure and to a lesser degree with age, and FEV_1 appears to relate to occupational exposure as well as smoking. As no decline in FEV_1/FVC is noted in non-smokers, the authors felt that smoking had the more important effect. The increased incidence of bronchitis in non-smokers and former smokers over age 55 lends additional support to the importance of the occupational factor, as well as smoking. (It was not possible to conclude from this study whether or not an unusual incidence of chronic bronchitis existed among persons who worked only underground at Denison Mines where diesel equipment had been used.)

The commission on the health and safety of workers in mines noted that there was persistent evidence from other jurisdictions that miners exposed to dust have more respiratory disability than the comparison groups studied and that the effects were related to dose. The report concluded, however, that the separate effects of mine contaminants, smoking, air pollution and socio-economic factors on the prevalence of chronic bronchitis among hardrock miners was not clearly established and suggested more extensive epidemiological research on the prevalence of chronic bronchitis in Ontario mines and plants. It also pointed to the need for further epidemiological review of possible effects from the use of diesel engines, which began in Ontario about 1950 and are now widely used.[83] Meanwhile, no compensation for chronic obstructive lung disease is presently granted to miners in Elliot Lake or elsewhere in Ontario, as will be discussed in Part B.

Other Studies on Industrial Bronchitis: While the role of cigarette smoking in the development of bronchitis has been well-documented for many years, the influence of industrial exposure to dust and fumes has been less well accepted. Several recent investigations have detected an increased risk of developing of bronchitis in certain occupations, however, the significance of the occupational exposure has been downplayed when compared with smoking. The precise relationship between smoking and job is not well established in most of these studies and consequently the issue remains controversial. As pointed out by Weill and Diem,[84] major alternatives for these relationships include the possibility that because smoking has a powerful influence on the production of chronic bronchitis, it might mask the occupational dose-response relationships in smokers. Alternatively, they state that smoking and dust or other exposures might interact synergistically to enhance the dose-response relationship. Both possibilities confound the issues of sorting out work-relatedness.

A study correlating years of foundry work to bronchitis rates, claims that working on the foundry floor increases the prevalence of bronchitis by about 60% after accounting for smoking habits. Non-smoking foundry workers appeared to have the same prevalence of bronchitis at age 60 as controls (non-exposed workers) who were heavy cigarette smokers.[85] Another study comparing 41 core workers from a cast iron foundry with 45 workers from the same foundry apparently found that clinical manifestations of chronic bronchitis appeared to be significantly more common in the core worker group than among the controls, with a difference in pulmonary function abnormalities also suggested.[86] Joosting estimated the impact of smoking as well as dust exposure in iron and steel foundry workers in terms of loss of pulmonary function equivalent to number of years of accelerated aging. At exposure in the order of 2.5 mg/m^3 of fine dust the mean loss of FEV_1 and FVC was about 7-10 functional years. In contrast, the "age-loss" attributed to smoking in this study was calculated as 1-4 years in moderate to heavy smokers. (The methodology in this report is admittedly questionable, and as the standard errors were large the author cautions that the predictability of readings in individual cases is minimal.)[87]

A number of studies on coal miners, both in Britiain and in the U.S., have documented an increased incidence of bronchitis in this occupation. Hankinson et al. showed that expiratory flow rates indicative of airway obstruction were indeed decreased with age, smoking and years of dust exposure, independently of radiological category of coal workers' pneumoconiosis. They have therefore concluded that industrial bronchitis does indeed occur in American coal mines.[88] Rogan et al. also documented this increase in industrial bronchitis in a massive and often cited study of British coal miners; but, again, they stressed the effect of cigarette smoking as a large contributor to this disease.[89] Their conclusions echo those of others with respect to industrial bronchitis in coal mines.[90] Some authors have noted, that indeed there is a definite relationship between the prevalence of bronchitis and the level of dust exposure,[91] although it has been pointed out that the dust that is usually measured and that which causes the abnormal x-rays, i.e. the respirable fraction, is not the same dust in terms of particle size as the dust that causes the airways disease.[92] Recently published results of a 9 year follow-up of the population of two U.S. mining towns have again shown a higher prevalence of symptoms and lower mean FEV_1 in miners and ex-miners compared with non-miners. Proportional mortality rates among miners and ex-miners for respiratory diseases and for accidents were higher than among non-miners. Miners also had a high SMR for non-malignant respiratory disease. It was concluded that cigarette smoking and mining exert independent effects.[93]

Morgan has pointed out that it is clearly important to define the role of industrial bronchitis in the pathogenesis of occupationally-related lung disease as first, it seems that the increased standardized mortality ratio in various dusty occupations is a result of chronic bronchitis; secondly, that the etiology of the pneumoconioses and bronchitis are different, and thirdly, because most preventive measures and, in particular, dust standards are directed at the control of pneumoconioses rather than of industrial bronchitis and consequently, present dust control methods might well prove to be ineffective or deficient with respect to the overall problem of occupational lung disease. He also commented in his discussion of the coal miners' studies that cigarette smoking has about a 5 to 10 times greater effect on ventilatory capacity than does coal dust.[94] Although this figure has been widely quoted (specifically by Workers' Compensation Board personnel), the basis of it remains most unclear, especially considering that appropriate dust measurements were never available. Moreover, one of the authors of the British coal miners study specifically objected to the conclusion that smoking is the predominant factor, pointing out explicitly that the data do not support this claim.[95] In a more refined study of lung function in coal miners (longitudinal analysis of FEV_1 was calculated from the results of serial cross-sectional epidemiological surveys over about 11 years), Love and Miller[96] again confirmed that FEV_1 loss increased with cumulative dust exposure. They found that lung function loss attributable to average dust exposure amounted to about one-third of the loss attributable to smoking, but that at high dust exposures the effect was similar to that of smoking. Most interestingly, they found that only about 7% of the variance could be explained by differences in age, height, smoking and dust, suggesting that the average predicted response is a poor guide to the likely response of an individual.

Other studies that have linked mineral dust exposures with increased prevalence of bronchitis include MacDonald's report about asbestos miners in Quebec,[97] and Sluis-Cramer's studies of gold miners in South Africa (where the excess of bronchitis in occupationally exposed workers compared to non-industrially-exposed workers was found only in smokers).[98] Walker et al. found that bronchitis was more prevalent in coke oven workers than in workers in other areas of the industry, but again the authors note the stronger association between bronchitis and cigarette smoking than that between bronchitis and industrial exposure.[99] Chronic bronchitis has also been found to be increased in workers exposed to organic dusts. Merchant et al. found a strongly significant interaction between smoking and dust exposure in their effect upon prevalence of byssinosis and chronic bronchitis as well as declining ventilatory function.[100] Bouhuys and Zuskins however found that the decline in FEV_1 was more marked in non-smoking hemp workers as compared to controls, than among smokers.[101] Workers in the chemical industry also appear to have an increased incidence of chronic bronchitis[102] and increased morbidity with regard to chronic bronchitis and chronic obstructive lung disease has been observed in the rubber industry.[103] In a major study of the gas industry, Doll found an increased mortality from bronchitis in some workers in the process of gas production from coal, however, he concluded that even among workers exposed to high concentrations of sulphur dioxide, the "industrial component" of bronchitis was quite small.[104]

Other studies have shown a high incidence of chronic bronchitis among welders,[105] and among workers in the cement industry and aluminum plants.[106] While some authors found evidence linking exposure to resin systems (phenol-formaldehyde) with chronic airway disease,[107] other authors found no evidence of chronic airways obstruction in workers exposed to polyester resins.[108] Bates has concluded that most, if not all, occupations involving heavy dust exposure carry an increased morbidity from chronic bronchitis.[109]

As alluded to in the previous sections of this chapter, the various diseases in the chronic obstructive lung disease group are not always well-defined. Morgan and others have pointed out that the disease characterized by cough and sputum for at least three months a year (i.e. chronic bronchitis) may or may not be associated with airway obstruction. While it is generally agreed that dust effects can not really be separated from those of cigarette smoking in an individual patient, population studies have shown that the response to tobacco smoke may in fact be different from that caused by industrial dust.[110] Morgan has even taken issue with the conclusion in 1966 of the British Medical Research Council that "it is not possible now, nor likely to be possible in the future, to determine in any particular individual with chronic bronchitis how much of his or her illness is attributable to any particular environmental factor."[111] He feels that unlike bronchitis induced by cigarette smoke, the predominate effect of industrial bronchitis is on the larger rather than smaller airways and the condition is not accompanied by emphysema.[112] (As discussed in the above section, the common pulmonary function tests such as FEV_1 reflect mainly the behaviour of the upper airways while flow rates at 75% of vital capacity or FEF 75 and beyond, are governed mainly by the state of the smaller airways.) Morgan states that the evidence indicates that in a non-smoking individual with long continuous dust exposure who has a cough and sputum plus a minor degree of large airway obstruction, both the symptoms and the pulmonary impairment can be assumed to be a consequence of occupational factors.[113]

It can thus be concluded that much of the reduction in ventilatory function that has been observed in dusty trades is a reflection of industrial bronchitis rather than the pneumoconiotic process as revealed by x-ray opacities. There seems to be a general consensus that in a non-smoker in a dusty environment, a decline in FEV_1 should be assumed to be job-related; even in a smoker, severe decline in FEV_1 especially in the absence of increased TLC(indicative of emphysema), is suggestive of dust-induced damage.

Although precise quantification of the scope of the occupational respiratory problem is a long way off, it is abundantly clear that the problem is of no small magnitude, and as will be shown, reaches well beyond that reflected by current workers' compensation statistics.

4. Issues of Diagnostic Accuracy and Disability Assessments

4.1 Evaluation of Impairment and Disability Attributable to Respiratory Disease

Impairment and Disability: As discussed in Section 2 above, the ways in which the respiratory system may be damaged are diverse and there is indeed overlap amongst the different diseases. Although improvements in laboratory methods have done much to enhance the accuracy of diagnosis and the evaluation of therapeutic effectiveness these increasingly sophisticated diagnostic tools and the greater appreciation of the enormous reserve of the body has made it more and more difficult to establish a frontier between health and disease.

Nevertheless, physicians have been increasingly asked to quantify the seriousness of a disease, so that administrative decisions may be based upon it. It is recognized that a permanent impairment of the respiratory system is not generally static; and periodic re-evaluations at appropriate intervals are often necessary. Moreover, it is well-recognized that the measurable degree of dysfunction of the respiratory system does not necessarily parallel either the extent and severity of the anatomic changes of the lungs or the patient's own account of his or her difficulties in carrying out daily activities. Nevertheless, compensation is a necessity as a consequence of society's inability to guarantee a risk-free environment and hence some means of assessing impairment and/or disability is also necessary.

Determination of work-relatedness depends heavily on the accuracy of diagnosis, which in turn is based largely on the interpretation of the various parameters available to the physician. The precision of these parameters has even greater significance in the evaluation of the extent of disability, both in terms of their use in quantifying disability payments as well as in the determination of an individual's work capacity. The latter issues are not only a frequent source of controversy within workers' compensation systems, but remain problematic in any social security system where payment is determined by some measure of disability. An understanding of the issues relating to diagnostic accuracy and disability assessment is therefore of paramount importance.

Although there is considerable use of information on health "impairment" in "disability" decisions, it is necessary to distinguish between these two terms. The term "impairment" usually refers to some reduction of body organ function; "disability" indicates a lack of ability for a certain level or specific type of performance. For compensation purposes, the inability to work or to do a certain type of work is usually the primary concern. Clearly an individual may have a significant impairment but still be able to maintain his or her job. Conversely, an individual may be unable to continue at a particular job with only a minor impairment.

The Committee on Disability Criteria of the American Lung Association has discussed these terms in a document recently produced for use by organizations responsible for determining disability compensation.[114] "Impairment", they point out, "is purely a medical condition reflecting a functional abnormality which persists after appropriate therapy and with no reasonable prospect of improvement." It may be noted that this differs from the World Health Organization (WHO) definition which does not require the impairment to reflect a permanent abnormality--see Chapter I, Part A-2.2. There is agreement, though, that "disability" is a term which indicates the total effect of impairment upon a person's life and is affected by such diverse factors as age, gender, education, economic and social environment, as well as energy requirements of the occupation. The American Lung Association reiterated the increasingly accepted belief that rating of health impairment falls within the province of a physician's expertise to measure, however determination of disability requires consideration of many non-medical as well as medical variables and is thus an administrative decision.

The Canadian Task Force applied these concepts with respect to respiratory disease defining "impairment" as a reduction of lung function as compared with predicted so-called "normal" values. "Disability", they defined as the inability of an individual to perform his/her usual activities at work, at home or in his/her leisure time. They also stressed that the criteria for impairment and disability should not be confused with diagnostic criteria, although some overlap may exist.[115]

Gaensler and Wright, in their often cited treatise on the subject, point out that an evaluation of the capacity to perform can be made and expressed in terms of what the person can actually do. In contrast, recognition of impairment implies that the capacity for a specific function has become less than that previously possessed. Since ordinarily the physician has no personal knowledge of the examinee's capacity to perform at an earlier time, the approach by necessity has to be that of comparing the person to a statistical figure, namely, the "normal" man or woman.[116] Because of the wide variations of "normal" this method of assessment must be undertaken cautiously.

Parameters for identifying impairment: The parameters used by the physician for identifying impairment of the respiratory system consist of the medical history, the physical examination, chest x-ray and pulmonary function tests.

The medical history includes the gathering of demographic information about the patient (including age, gender, race, marital status, education and lifestyle) and clinical information such as specifics regarding breathlessness, wheezing, cough, sputum production, pain, frequency of respiratory infections, as well as the present or past history of other respiratory conditions and allergies. It also includes the gathering of detailed information regarding employment background and environmental exposures, as well as hobbies, pets and use of tobacco. The difficulties in assessment of "dyspnea" or breathlessness relates to its subjective nature, as will be alluded to below. Even more difficult to assess but nonetheless important, are the symptoms related to the worker's general well-being such as fatigueability, weakness and loss of appetite.

The physical examination includes general observations of the patient's chest movements, muscle wasting, pattern of breathing, the presence of cyanosis (a bluish discolouration of the mouth and lips), clubbing of the fingers, as well as evaluation of the character of the lung sounds, wheezing, crackles and so on. Also, evidence of cor pulmonale (right heart failure) is sought. There is a large variability in assessment of these parameters by different clinicians and it is generally agreed that physical examination is a fairly crude method of evaluation.

While chest x-rays are essential in establishing the diagnosis in many conditions and may contribute to the overall assessment especially with respect to progression of findings, it is now widely recognized that the correlation between x-ray findings and respiratory impairment is poor. As was discussed in the preceding section, there are many examples of demonstrable functional impairment without radiological changes, and radiological findings without measurable functional impairment. For example, the x-ray changes of coal workers' pneumoconiosis and simple silicosis are often marked even before there is measurable impairment of lung function. However, in asbestosis, there is a closer relationship between x-ray abnormalities and impairment of function and it is possible that some lung function disturbances can precede recognizable x-ray changes. In advanced cases of asbestosis, gas exchange may continue to deteriorate while the chest x-ray remains unchanged. Also, there may be considerable variation of functional impairment in the presence of relatively little radiological change, for example, in chronic bronchitis. The variability in interpretation of x-ray findings will also be discussed below as it is frequently an issue in compensation battles. Both the American Lung Assocation and the Canadian Task Force on Occupational Respiratory Diseases (Pneumoconioses) strongly recommend the use of the International Code (ILO U/C) for pneumoconiosis classification in describing all types of diffuse interstitial lung disease. The Canadian Task Force recommends that a minimum of category 1 changes be required for a positive radiological diagnosis.[117] Although this ILO U/C classification system is used worldwide, Ontario is one of the few remaining places not to have yet changed over to this method.

Pulmonary function tests are the cornerstone of assessment of respiratory impairment. A prevalent opinion is that it is erroneous to adopt sophisticated techniques capable of detecting minor deviations from "normal", if these have little or no consequence on the performance or lifestyle of the worker. Ideally, pulmonary function test results should be compared to pre-exposure data on the same individual. Pre-employment medical examinations and their use and abuse are the subject of much controversy. Furthermore, information obtained in these examinations is often unavailable to those suffering the effects of previous employment in hazardous conditions. Thus at the present time assessments of pulmonary function must generally be made without data to indicate if a reduction greater than that which would be accounted for by aging had even occurred.

The American Lung Association proposed a test scheme for determining if an individual is severely impaired by respiratory disease based on a progression of tests as shown diagramatically in Figure 6.[118] The first step consists of assessing ventilation by the spirometric measurements of FVC and FEV_1 (as explained in Section 2). The second step consists of assessing the diffusion capacity of the lungs. The third step consists of estimating the individual's work capacity through the use of a simple clinical exercise test as described by Jones.[119] (The maximum oxygen uptake is calculated by determining the maximum obtainable power output on either an ergonometer or a treadmill and measuring the heart rate and ventilation during exercise. The Committee concluded that a claimant should be considered severely impaired if 30% to 40% of his or her observed oxygen consumption at maximum tolerated work rate (VO_2 max) does not allow the claimant to meet the oxygen cost of his or her occupational activities over an eight-hour period. If VO_2 max of 15 milligrams per kilogram per minute cannot be reached, the participant should be considered impaired for practically all types of labour.) The Committee suggests that the fourth step of this scheme be performed on claimants on whom there is still uncertainty. They list the following as a basis for doubt: inappropriate changes in heart rate and/or expired volume of air per minute, subject unable to complete the test because of non-respiratory systems (for example leg muscle fatigue) and doubt about the predominant factor responsible for the respiratory symptoms. In this procedure, in addition to recording the usual parameters, arterial blood specimens are taken to assess cardiac output, alveolar ventilation and physiological dead space per tidal-volume ratio (see Section 2). (No recommendations were provided in their document with respect to assessment of moderate or mild respiratory impairment, presumably because partial disability awards are not commonly found in the U.S. disability schemes, or are based on wage-loss replacement rather than on percent impairment.)

Rating of Impairment: As will be discussed in Part B of this chapter, compensation awards in Ontario, are currently based on the extent of impairment. The relationship between the results of pulmonary function tests and impairment is not linear and it would clearly be an oversimplification to say that a 30% reduction of the vital capacity equals 30% impairment. For this reason, the overall assessment of the worker has to be surveyed. No table officially exists to guide the Ontario WCB in its assessment of respiratory disease. Although tables can serve only as a rough guide, it is informative to examine the different rating systems that have been published in other jurisdictions.

As discussed above, the pneumoconioses generally produce a restrictive pattern on lung function tests although it is possible for some obstructive features to be present as well, caused directly by the pneumoconiotic process or by an associated chronic obstructive lung disease. As the difficulty in assessing the amount of impairment caused exclusively by pneumoconioses is confounded by airway obstruction often attributable to cigarette smoking or to other occupational causes (dusts, fumes, etc.), some classification systems consider the degree of impairment under two headings: restrictive and obstructive patterns. Forty to 60% of predicted values for FVC (restrictive) or FEV_1 (obstructive) is considered moderate impairment, less than 40% is considered severe, more than 60% but less than "normal" is considered mild impairment.[120]

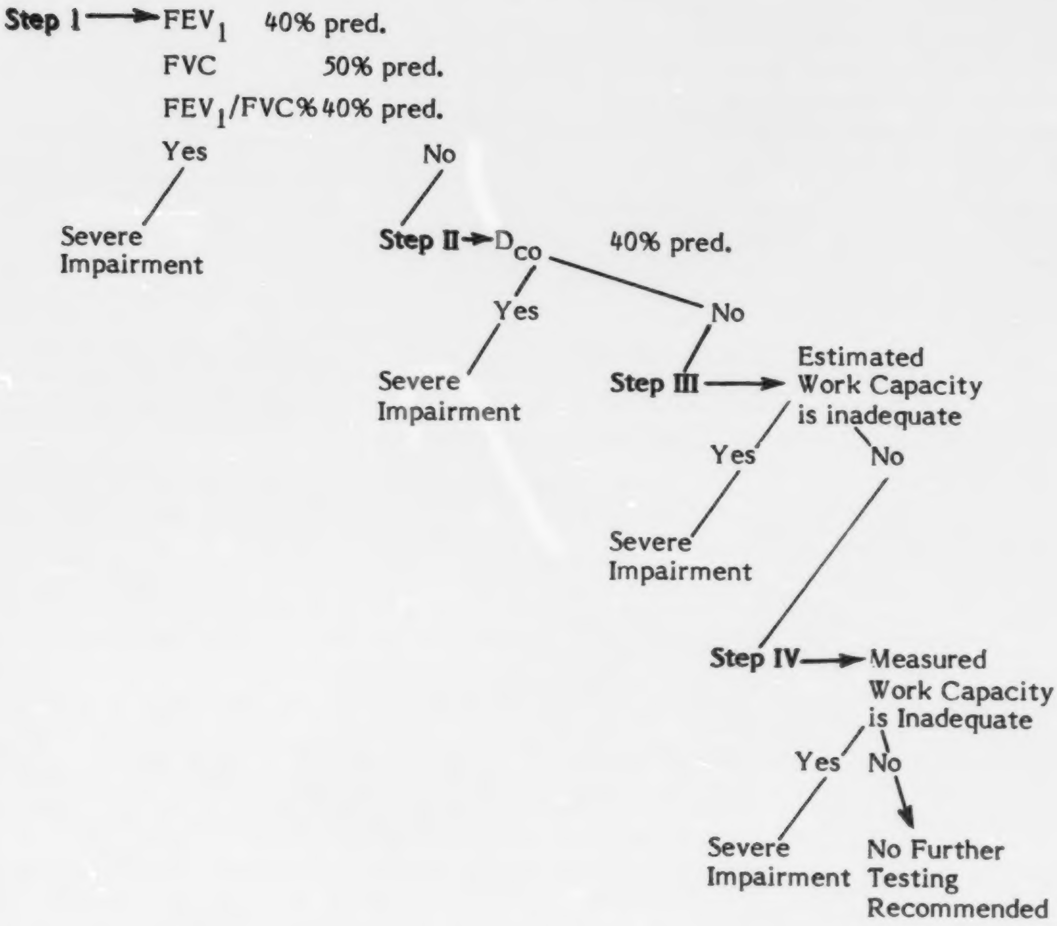
The full classification of impairment proposed by the Canadian Task Force is provided in Appendix 9. It is also noted that the degree of dyspnea often does not correlate with the abnormal results of lung function tests within the same class and that factors such as age, concomitant illness, physical fitness, weight and smoking habits must be taken into account. It is pointed out that a history of smoking may seriously compromise the ability to evaluate respiratory impairment specifically attributable to pneumoconiosis.[121] The chart developed by Gaensler and Wright in 1966 corresponds closely to the recommendations of the Task Force. These authors noted that the subdivisions between obstructive impairments are relatively simple compared to the much more difficult divisions in the restrictive group wherein the transition from minimal to total disability may be abrupt.[122]

In a study carried out in 1979, Epler and co-workers found that the relationship of dyspnea to physical data was much better for obstructive diseases than for restrictive ones.[123] Their observations, however, reiterated the conclusion that the overall correlation between subjective symptoms and objective lung impairment is poor and hence the history and clinical examination cannot be relied upon alone. They compared their rating scheme with those published by five other groups with respect to criteria indicative of "total disability": The U.S. Social Security Administration, being concerned only with permanent and total disability, published upper limits for MVV (maximum voluntary ventilation--the volume of air in litres per minute exhaled in a specified period during repetitive maximum effort), FVC and FEV_1 based on height alone.[124] The Veterans Administration specified dyspnea of grades 3 and 4 as indicative of 60% to 100% impairment.[125] The Committee on Ratings of the American Lung Association considered 60% to 90% as equivalent to severe impairment. To qualify as severely impaired in their rating scheme, any two of FVC, FEV_1 or MVV must be less than 55% predicted.[126] Wilson and co-workers published complex formulae for calculating the degree of pulmonary disability.[127] The amended Federal Mines, Safety and Health (Coal Miners) Act of 1977 reassigned disability adjudication to the Department of Labour which proposed limits of FEV_1 and MVV that were presumed to be more lenient than those of the Social Security Administration.[128] A table was presented by Epler et al summarizing the differences in the U.S. rating schemes. An adaptation of this table is presented in Appendix 9 along with the Canadian Task Force's recommended scheme.

Epler et al. calculated the percentage of their patients with diffuse infiltrative disease and the percentage of asbestos-exposed workers who would be classified as severely impaired using each of the six U.S. schemes. They found that their scheme and that of Wilson et al resulted in nearly the same percentage in each group, whereas the stricter limit of the Social Security Administration selected fewer persons. The new criteria for coal miners failed to have the intended more liberal results with interstitial disease. The authors point out that this is due to the fact that both FEV_1 and MVV must be below the specified limits, and patients with interstitial pulmonary disease, unlike those with airflow obstruction, often have well preserved MVVs and therefore do not qualify. They also pointed out that if the wording were changed to FEV_1 or MVV, then a much greater percentage of the patients would qualify. It was not possible to tell whether patients identified as severely disabled by one scheme were similarily identified by the others. However, by including only the 37 patients who were followed at least three years prior to death, the Social Security criterion of FVC of 40% predicted failed to select 32 (86.5%), whereas the most liberal AMA standard of FVC of less than 55% predicted, missed 12. The complete Social Security criteria of FVC less than 40% predicted or DCB (single breath diffusing capacity) less than 30% were still too strict: almost 50% (15 of 37) were not

Fig. IIIA-8

Test Schema - Determining Severe Respiratory Impairment


Step I → FEV_1 40% pred.
FVC 50% pred.
FEV_1/FVC% 40% pred.
Yes
No
Severe Impairment
Step II → D_{co} 40% pred.
Yes
No
Severe Impairment
Step III → Estimated Work Capacity is inadequate
Yes
No
Severe Impairment
Step IV → Measured Work Capacity is Inadequate
Yes
No
Severe Impairment
No Further Testing Recommended

Source: Committee on Disability Criteria of the American Lung Association, American Thoracic Society News, Summer 1981.

considered totally disabled. The authors note that their own criteria failed to identify as severely disabled only 3 of these 37. Although it is noted that mortality data do not necessarily correlate with evaluation of impairment from many diseases, they felt that the evidence in chronic pulmonary disease was sufficient to establish a relationship between severity of functional impairment and survival.

Thus the accuracy with which a rating scheme can predict actual severity of respiratory disease is quite variable. Despite the evidence presented as to the superiority of one system in this regard, the lack of consensus as to the best method to rate disability is itself quite telling.

The approach used in some countries entails the use of all tests of lung function ever devised, with the report of total functional profile analyzed by computer.[129] This approach has been largely rejected on the basis that it would lead to many traumatic, painful and expensive tests being performed unnecessarily.[130]

As x-ray abnormalities have received much attention in the pneumoconioses, Epler et al. also compared the ILO radiological ratings in their patients with the histological or physiological abnormalities and confirmed the observation that x-ray severity correlates poorly with functional impairment. Among severely impaired patients, only approximately half had severe x-ray findings. They explained that the problem is that the linear or irregular opacities due to asbestosis, for example, are similar to markings normally present, therefore the linear shadows are often not recognized until late in the disease and the functional impairment may be underestimated. (By contrast, it is pointed out that the rounded opacities of silicosis, for example, are not normally seen and therefore, are more easily recognized.) Furthermore, they point out that impairment is related not so much to the number and size of nodules as to the conditions of the surrounding interstitial tissue, as mentioned in earlier sections. Hence, the conclusion of many groups including the Task Force on Respiratory Diseases (Pneumoconioses) that "a chest x-ray is unsuitable for estimating the degree of pulmonary impairment" was reiterated.[131]

The next three sections will elaborate further on some aspects of the three issues identified in this study: the problems of reliance on chest x-ray readings, limitations in the interpretation of pulmonary function tests and lastly, and most important, issues relating to the correlation of impairment with disability.

4.2 *Chest X-Rays: Intra- and Inter-Reader Variability*

Although many studies have shown and many groups agree that chest x-rays do not correlate well with functional respiratory impairment, radiology remains of paramount importance in establishing the diagnosis in pneumoconiosis. However much controversy is generated by chest x-ray interpretations in the Ontario workers' compensation system. Quite apart from the issue of lack of correlation between chest x-ray findings and other parameters is the fact that there is considerable divergence of opinion among different readers of a chest x-ray as to its interpretation.

One study using experienced chest x-ray readers clearly illustrated this reality: A total of 2,337 radiographs were interpreted independently by the three experienced radiologists who constituted the U.S. Public Health Service panel. In addition, the films were interpreted by an epidemiologist with extensive experience in the radiological aspects of coal workers' pneumoconiosis. The films were categorized according to the ILO radiological classification, however, to simplify the study the finer differentiations were ignored and only four basic categories were considered: no pneumoconiosis, suspect, simple and complicated pneumoconiosis. In only slightly more than half the films was there agreement among the four film readers. A wide divergence, i.e. a difference of two or more increments existed in greater than 10% of the films. The authors felt that this divergence of opinion was highly significant, especially in that the divergence of opinion was not merely a disparity between the subdivisions. Moreover, in almost 20% of the films there was divergence of opinion on whether or not complicated silicosis was present. Although they felt that the extent of inter-observer error is greater than generally appreciated, the authors stressed that this should not invalidate the uses of radiology to assess the category of the disease; but should highlight the fact that the categorization even by an experienced observer is not absolute.[132]

Other studies regarding the accuracy of diagnosis of tuberculosis have noted even greater reader variability in assessing chest x-rays, and in fact, that the same observer might not place the film in the same category on a second occasion.[133] For example, 4 sets of x-ray films of 1,256 people were each interpreted twice by 5 experienced radiologists. On one set of films the number of positive diagnoses ranged from 56 to 100. Moreover, the 56 chosen by one radiologist were not all included in the 100 chosen by another. Similarly each reader disagreed with his own readings when he interpreted the same film a second time. For example, one reader who interpreted 59 films as positive when he read them the first time called as many as 78 positive the second time. Here again the 78 did not include all the 59. The same was true for the other readers.[134] Another study, conducted on 1,800 x-rays submitted for independent interpretation by 6 readers, showed that disagreements on x-rays called "positive" by at least 1 of them occurred in 74% i.e. on 69 of the 93 films thought to be positive by at least 1 radiologist.[135]

Also of interest is the series of studies that show that people not experienced in the radiology of pneumoconioses can be trained to classify chest radiographs (at least for epidemiological purposes) with as much accuracy as experienced radiographers. In one study, 16 people (including 8 non-medical) inexperienced in the radiology of occupational lung diseases classified 300 selected chest x-rays, of which 220 had been previously classified by eight experienced medical readers. Variability among readers was greater in the experimental panels than among the experienced readers, but the average consistency between pairs of novices and their use of the ILO categories for the same radiograph was similar (about 29%) to the average consistency among the experienced readers. The authors pointed out that the success of such experiments illustrates that classification of chest x-rays is purely a descriptive tool.[136]

Thus, not only do chest x-rays not necessarily correlate with severity of disease, but there is considerable variability in interpretation between readers as well as inconsistencies even in the same reader's interpretations. This should be borne in mind in light of the role the chest x-ray often plays in determining eligibility for compensation as well as percentage of disability award.

As Yerushalmy has pointed out[137], as discouraging as the results of interpreting single x-ray films are, even more shocking is the fact that the interpretation of serial x-rays is also grossly inconsistent. A comprehensive analysis of this issue, in which almost 10,000 chest x-ray comparisons were made [138], indicated that two readers are likely to disagee with each other concerning evidence of progression, regression or stability of disease in approximately one third of the cases, and a single reader is likely to disagree with himself in about one-fifth of the film pairs. As it seems that the progression of pneumouniosis is often defined largely in terms of comparison of the features in two or more films taken over a period, these findings may have important implications for workers' compensation.

4.3 *Limitations in the Interpretation of Pulmonary Function Tests*

Aside from the variable weighting of the different pulmonary function tests in different impairment rating schemes, there are certain inherent limitations in the interpretation of pulmonary function tests that need to be addressed.

First, the standard pulmonary function tests (FEV_1, FVC) require maximum voluntary effort from the patient, who for various reasons may be unable or reluctant to perform the task as well as his or her ventilatory capacity permits. Reasons for this include possible lack of understanding of the test, the state of physical training, fear of cough, chest pains or shortness of breath, effects of other illnesses particularly heart disease, the effects of temporary factors such as presence of a respiratory infection, or factors related to the patient's motivation and co-operation. By recording the flow volume curves (from which flow rates, such as FEF25-75 or FEF75-85 can be calculated), erroneous results brought about by improper or incomplete performance can, nonetheless, be detected and these newer techniques are being increasingly used. However, while these latter tests minimize the subjectivity factor of the standard pulmonary function tests, their results are less reproducible, i.e. there is greater variability in the actual measurement.

Secondly, the tests may fail to detect significant respiratory impairment if the ventilation function of the lungs is relatively normal even though there may be serious impairment in gas exchange. The reasons for this are evident from the discussion of pulmonary physiology in Section 2 in which it was explained that the diffusion and perfusion functions are somewhat distinct from the ventilation function. (Although usually patients with impaired gas exchange do have some reduction of vital capacity.)

Thirdly, the variation of "normal" among people of the same sex, age and height is considerable. The questions that need to be addressed include the following: What is a "normal" population? Which normal values should be used as a comparison for a given individual? and Does "beneath the lower limit of normal" necessarily mean "abnormal"? Here the influence of age, race and other factors on spirometric results must be considered. Decisions with respect to establishing the lower limits of "normal" especially in the interpretation of the percentage predicted are important in assessing impairment. (These issues are discussed at length in a recent review article by Miller and Thornton.[139])

Even if the tests were completely unaffected by effort factors, were consistently reproducible, were accurately reflective of the extent of disease and were based on comparison to a well-matched standard, the meaning of the result might still be questionable. Cary and co-workers noted that the descriptive terms used by physicians in the interpretation of pulmonary function test results can have important consequences in terms of a patient's employability and eligibility for compensation. They therefore conducted a study by asking the 26 pulmonary physicians from the respiratory disease staff of their university and practicing chest physicians in the community to interpret results of ten consecutive pulmonary function tests from one lab. They found frequent disagreement in their assessments of respiratory impairment, as summarized in Table A in Appendix 10. They noted that the different sets of published criteria for both obstructive and restrictive disease categories apply different numerical limits to the descriptions of "normal", "mild", "moderate" and "severe". (Different interpretations of reduction in FEV_1/VC ratio are shown in Table B of Appendix 10.) The authors suggest that as long as standards for defining the presence and severity of respiratory disease are arbitrary, there will be disagreement between physicians concerning the interpretation and description of pulmonary function data.[140]

Thus, what one physician considers severely disabled may be considered only moderately or even mildly disabled by other physicians. Herein lies a significant problem for compensation systems.

4.4 *"Subjective" Symptoms and "Objective" Testing: Correlation of Impairment and Disability Assessments*

One of the major sources of controversy within the compensation system stems from the reality that an individual's feeling of disablement is not always reflected in the results of the various tests and hence the worker is dissatisfied that his or her rating does not truly reflect his or her disability. Although it is generally acknowledged that impairment and disability are not synonymous and that only the former falls within the domain of the medical professional, the subjective symptoms of the patient are usually said to be taken into account by the physician in arriving at an assessment.

The feeling of breathlessness (dyspnea) is a sensory experience which like pain can be perceived only by the patient. It implies consciousness of breathing effort or feeling of air hunger, which in the patient's opinion arises at a level of exertion less than that at which it is normally encountered. Voicing the complaint of shortness of breath requires first an awareness of the sensation and secondly, an opinion that this sensation is inappropriate, unpleasant or disabling. Factors other than the presence or degree of organic disease may modify both these factors. The list includes difficulty of verbal expression, degree of training, judgment expressed by co-workers and relatives, the reaction to unaccustomed or unpleasant bodily sensations and the worker's opinion concerning "normal limitations" as a result of aging. Moreover, many people never experience dypsnea despite considerable pulmonary impairment, because the exertion required for their daily tasks, both occupational and recreational, and the vigor and speed with which they are customarily performed is slight. A manual labourer might thus be disabled by mild pulmonary impairment, while an executive may complain of little disability despite severe impairment.[141]

Several studies concerning individuals who claim disability due to respiratory disease showed that the one variable which related most closely to work status was not impaired lung function but socio-economic class of the claimant.[142] One study confirmed that social class factors were related to disability primarily through correlation with occupational difficulty with no significant independent effects of "social class", intelligence or school grade achieved when occupational difficulty was held constant.[143] This might seem intuitively obvious--the harder the job the higher the relative disability. However, this reality is often blurred by other factors which have also been purported to play a role, namely, the "personality factors". One study claims to show that three personality scales (the Minnestoa Multi-phasic Personality Inventory, the Hysteria Psychopathic Deviate, and Masculinity-Feminity Scales) can account for a large proportion of the variance in work status that was not accounted for by the severity of the patient's physiological impairment and exertional demands of the patient's occupation.[144]

Perhaps more importantly, it is now well-established that physical impairment may itself induce psychological impairment. It has been shown that the majority of patients who suffer from chronic progressive respiratory disability suffer depression or anxiety or both, which may be manifested by sadness, lack of motivation, sense of worthlessness and even thoughts about suicide.[145] One study indeed showed that 17 of 23 patients with chronic obstructive lung disease who entered a rehabilitation program were noted to have some "crippling" degree of depression.[146] As there are many variables that may influence an individual's psychological response to the stress of physical illness [147], it is certainly not surprising that "objective" tests of physical impairment may be inadequate indicators of disability.

In summary, "disability" and "impairment" refer to different concepts, although impairment assessments often play a role in determining eligibility and level of disability pensions. Only the assessment of impairment is a medical matter. It, in turn, is based upon interpretations of the medical history of the individual, the physical examination, chest x-ray and pulmonary function tests. History and physical examination are felt to be an inadequate basis for assessment; chest x-rays are important in diagnosis of many conditions, but generally correlate poorly with symptoms or pulmonary function tests and are subject to reader variability in interpretation; pulmonary function tests form the cornerstone of impairment assessments but there is no uniform standard on which the selection, weighting and interpretation of these tests is based. It is believed that the extent of disability correlates more strongly with the exertional demands of the individual's occupation than with objective measurements of impairment. Moreover psychological impairment often compounds the actual physical damage, making it ever more difficult to assess the "true" extent of disability.

How these variables presently come into play in compensation awards is discussed in Part B of this chapter. Although the disability assessments in this jurisdiction are made by physicians and are based on an impairment model, it is clearly untrue that decisions are made purely on objective measurements of physical impairment. Suffice it to say that various opinions on the weighting of the various factors are possible; the "best opinion" is dependent, in large part, on the philosophical underpinnings involved. The dual award system proposed by Ontario's White Paper on the Workers' Compensation Act offers a possible approach to resolving this issue by drawing a distinction between "impairment" and "disability" for purposes of calculating benefits. Although some oppose the provision of a lump sum for physical impairment in combination with payments for actual wage losses, preferring permanent and adjustable impairment pensions, the distinction clarifies the often muddled and controversial quantification of benefits, which, as will be shown in Part B has been the source of considerable discontent.

5. The Issues Summarized

1. Respiratory disease is a serious problem in Ontario representing a major cause of illness and suffering. The extent of occupational factors in respiratory diseases is largely unknown. Although some diseases have been recognized that result from specific agents that may be encountered in the workplace, such as silica or asbestos, many respiratory diseases are non-specific such as chronic bronchitis, emphysema or asthma, and may be caused by occupational as well as non-occupational factors. On reviewing the historical background of occupational respiratory disease, it is clear that the effects of industrial dust on ill-health have been known for at least four centuries.

   Difficulties in estimating the prevalence of occupational respiratory disease are related to the fact that there is no consensus as to what constitutes an "occupational" disease, the diagnostic criteria for each disease are not uniform, the number of workers exposed and the intensity of exposure to various hazards is largely unknown, dose-response data are lacking, and reporting systems are embryonic at best.

2. Respiratory disease is one of the five leading causes of death. Moreover, along with cancer, it is the only cause of death which is on the rise. Age-standardized mortality rates for bronchitis, emphysema and asthma have increased by 21% for males and 38% for females between 1969 and 1978, the crude death rate for emphysema with or without chronic bronchitis has risen faster during the last 15 years than any other cause of death, including lung cancer. Although smoking plays a major role, there is increasing evidence that occupational factors at least contribute to morbidity and mortality from this group of diseases. Thus, while it is difficult to appraise the dimensions of the occupational respiratory disease problem, this area of workers' compensation policy merits careful scrutiny. The structure and function of the respiratory system was reviewed to provide the reader with the basic knowledge needed to understand occupational lung diseases and the complexities in diagnosis and assessment. The factors determining ventilatory capacity were discussed in physiological terms, with explanations provided for the descriptions used in the measurement of lung volumes and flow rates. Many industrial lung diseases, particularly the pneumoconioses, effect the peripheral part of the lung; the pathology in the smaller airways is not easily detected by some of the standard pulmonary tests, and tests that are more sensitive to disease in this area of the lungs tend to be less reproducible.

The factors determining the diffusion of gas across the alveolar-capillary membrane were discussed, as well as the mechanisms whereby the lungs may become inadequately or inappropriately perfused with blood flow. It was shown how inadequate oxygenation may result due to scarring from asbestosis, for example, or due to *ventilation-perfusion imbalance that* may occur in severe obstructive lung disease for example. Oxygen deficiency may not only progress to respiratory failure, but even mild oxygen lack could reduce the healing potential of other organs of the body; respiratory disease could thereby contribute to morbidity and mortality from other conditions. The interrelation between cardiac and pulmonary disease was alluded to, specifically with respect to the development of cor pulmonale and the possible induction of cardiac arrhythmias by oxygen deficiency.

Dust is deposited in the lungs according to its aerodynamic properties largely reflected by the particle size. Monitoring and controlling dust in the air is currently largely aimed at eliminating small particles making up the "respirable fraction" from reaching its site of damage, the respiratory bronchioles and alveoli (the areas affected in the pneumoconioses). The dust that damages the airways is larger in size and is not, therefore, properly reflected in this fraction. This compounds the difficulty of assessing the role of workplace dust exposure in the development of chronic obstructive lung disease.

The normal mechanisms of lung protection were reviewed. Although the nose constitutes the first line of defence filtering out large particles, mouth breathing on exertion eliminates this route. The mucociliary escalator lining the airways is of paramount importance in the removal of particles from the lungs. It, however, may be rendered less efficient by exposure to toxic substances, cigarette smoke or other pollutants. Thus the presence of one toxic agent may increase the susceptibility of disease from other agents (including infection).

The mechanisms of removal of particles from the alveoli are poorly understood, but there is evidence that clearing of particles in this area is slower than for the larger airways which, in part, explains the increased effect of certain occupational hazards in this area.

Damage to the respiratory system may occur in many different ways depending in large part on the properties of the materials encountered. The inhaled substance may be an acute irritant evoking a short-lived, non-specific inflammatory reaction; it may be a low-grade irritant, evoking a non-specific chronic response, or it may serve to sensitize the airways or alveoli to produce an allergic lung reaction. Some substances have properties that act in more than one way. The mechanisms involved in these reactions have been discussed in this chapter. It was pointed out that while some mineral dusts may have irritant effects, others can cause fibrosis or scarring and some can even cause cancer. Mechanisms of pulmonary fibrosis and its variants were also discussed. It was pointed out that not only do different substances produce different effects, but that exposure in the real world is rarely pure and various combinations may manifest their effects in different ways. Individual variability will also modify the effects of the different agents. Thus, clinical presentation may differ markedly. Moreover, whereas the role of workplace factors in acute and dramatic disease may be readily apparent, the possible role of repeated but low level exposures in the development of a non-specific respiratory disease is much more difficult to assess.

Pneumoconioses, diseases produced by the inhalation of inorganic dusts, usually are associated with a "restrictive" ventilatory pattern on lung function tests indicative of a scarred, stiffened lung. It was noted, however, that although smoking is a major cause of chronic obstructive lung disease, it cannot be assumed that only the "restrictive" component of a disease is occupational in origin.

3. Section 3 reviewed the nature of occupational lung diseases prevalent in Ontario and alluded to the contentious issues with respect to compensation. It attempted to provide some indication of the incidence and severity of the known occupational respiratory diseases (i.e. the pneumoconioses), as well as to sketch out the possible dimensions of the role of workplace factors in the non-specific respiratory diseases.

Silicosis, caused by the inhalation of free silica, is the most important of the pneumoconioses in Ontario. Although simple silicosis is not a particularly disabling disease, many silicotics have chronic obstructive lung disease. Particularly in smokers, this complicates the task of sorting out the "occupational component" in the disability. X-ray changes in silicosis often appear early in the disease even before symptoms develop or changes in pulmonary function tests are evident. In some cases, silicosis may progress rapidly, in others, the impairment progresses insidiously. Silicosis may be complicated notably by chronic obstructive lung disease, but also by respiratory infections, particularly tuberculosis. Complicated silicosis is associated with considerable impairment, possibly leading to respiratory failure and cor pulmonale. There is no evidence that removal from exposure will halt or even slow the progression of the disease, hence, criteria for relocation and rehabilitation are controversial.

The history of studies with respect to silicosis in Ontario miners was reviewed, along with the statistics accumulated through the records of the Workers' Compensation Board. A Ministry of Health survey in the mid-1970s revealed that some 3.6% of Elliot Lake miners had silicosis and another 5.3% were pre-silicotic at the time of the survey. It was noted that although there are some indications that the severity of silicosis in Ontario miners is declining, this disease is still very much present. Almost 2,000 cases of silicosis have been recognized in Ontario to date.

Asbestosis, caused by the inhalation of asbestos, may be symptomatic even before radiological changes are noted. Unlike silicosis, this disease does tend to be characterized by the classical pulmonary function findings of a restrictive lung disease, and tends to be considerably disabling. It is unclear which parameters--clinical examination, x-ray findings or lung function tests are the most sensitive indicators of early disease, or which of the lung function tests is the most reliable. Although tuberculosis is not frequently associated with asbestosis, the complications of respiratory failure and cor pulmonale may develop. Asbestos-exposed workers have an increased risk of developing cancer as well, and their general life expectancy is reduced. Mortality studies of Ontario asbestos workers were specifically reviewed in this regard.

The efforts of the Task Force on Occupational Respiratory Disease (Pneumoconiosis) to ascertain the prevalence of pneumoconioses in Canada were reviewed. The deficiencies in the data base necessary for this endeavour were highlighted.

The nature of obstructive lung diseases was discussed. These diseases include chronic bronchitis, characterized by the production of cough and sputum; emphysema, characterized by the destruction of the alveolar spaces; and asthma, whose hallmark is reversible bronchial spasm. It was noted that cough and sputum do not necessarily imply airway obstruction and vice versa, however, many individuals with chronic bronchitis do indeed have obstructed airways causing them significant disability. Although it is generally agreed that differentiating the occupational from the smoking factor is difficult, some authors feel that certain features do distinguish industrial bronchitis from cigarette-induced bronchitis on the basis of location in the airways of the maximum damage. It has also been stated that in any non-smoker who works in a dusty environment and develops cough, sputum and mild airway obstruction, both the symptoms and pulmonary impairment should be considered work-related. Although the removal of a bronchitic worker from dust exposure may be beneficial in terms of reduction of cough and sputum and a slowing of the decrease in ventilatory capacity, the existence of some permanent damage is undeniable. The frequency with which chronic bronchitis progresses to emphysema, chronic respiratory failure or cor pulmonale is unknown.

In emphysema, there is actually a destruction of lung tissue. As its symptoms are those of airway obstruction, it is often difficult to make this diagnosis during life. However, some x-ray changes are characteristic as are some abnormalities in pulmonary function tests. The main issue with respect to compensation is the relative contribution of heredity, smoking and occupation. Apparently, 5% to 15% of the population have a particular predisposition to develop emphysema due to a genetic deficiency in alpha-1-antitrypsin. Oddly, this group has been singled out as eligible for compensation, as will be discussed below.

There are also some individuals who are predisposed to develop asthma, a disease characterized by reversible airway obstruction. Many occupational exposures can cause asthma, not only in those with this atopic predisposition, but in others as well. Diagnosis is made chiefly as a result of the characteristic history and temporal relationship between exposure and symptoms. The use of provocation tests to confirm work-relatedness is controversial as it may be dangerous and is not necessarily reliable. Once sensitization has occurred, the worker should no longer be exposed to that substance. Moreover, often a non-specific hypersensitivity develops which has significant effects on the workers' recreational activities as well as job potential. This has posed a problem from a compensation point of view.

Evidence suggesting that workplace conditions play a larger role in the chronic obstructive lung diseases than is currently recognized was drawn from a variety of sources. First, the multitude of studies linking specific occupational exposures to damage in the respiratory tract were alluded to. Secondly, inter-regional differences in chronic bronchitis and pulmonary impairment were discussed. The methodology and results indicating that the incidence of chronic bronchitis in Sudbury significantly exceeds that in Ottawa were presented. Thirdly, the studies revealing an increased incidence of chronic bronchitis in coal miners, foundry workers, asbestos miners, gold miners, coke oven workers, welders, hemp workers, chemical-industry workers and workers exposed to cotton dust were alluded to. The methodology and conclusions of an Ontario study of converter plant workers was presented, as was a study of Elliot Lake miners. The fourth source of evidence suggesting that occupational respiratory disease is underrecognized was the Discher survey, and a fifth source was the Survey of Disabled and Non-disabled Adults. It is therefore concluded that although smoking is the major cause of chronic obstructive lung disease, occupational exposures are certainly contributory. Moreover, without adequate exposure data, it is impossible to tell to what extent the workplace is responsible for the disease. Even with exposure data, sorting out the relative contribution of the workplace as opposed to smoking in any particular case is largely speculative.

4. Section 4 reviewed some of the issues regarding diagnostic accuracy and disability assessments. Impairment and disability are not synonymous. Whereas the former falls within the domain of the medical professional, the latter is dependent on a variety of non-medical factors and thus is an administrative decision. The measurable degree of dysfunction of the respiratory system fails to necessarily parallel the patient's own account of his or her disability. Moreover, measurable impairment does not necessarily reflect the full extent or severity of the anatomical changes of the lung. Not only are there variable systems of rating impairment, reflecting the lack of consensus as to the best method available, but the parameters themselves are subject to error. Further, there is considerable divergence of opinion as to what constitutes severe, moderate or mild impairment.

   The variability in chest x-ray interpretation that occurs when even expert radiologists assess the films was noted, as was the lack of consistency in x-ray rating even by the same individual. It was pointed out that chest x-rays do not correlate well with pulmonary function test results in any case. The limitations in interpretation of pulmonary function tests were also discussed especially with respect to deciding what constitutes a normal value. Studies have shown that the most important variable in assessing disability is the exertional demand of an occupation. This suggests that an accurate appraisal of the work-situation is as important in "disability" assessment as is the assessment of impairment. Lastly, the subjective or psychological impact of physical disability may be quite variable but is certainly an important consideration in victims of occupational respiratory disease.

   Thus, the task of establishing the work-relatedness of a respiratory condition, assessing the extent of disability, and distinguishing the occupationally-related disability from the non-occupationally-related are difficult and complex. Part B of this chapter will describe how these issues are dealt with by the workers' compensation system.

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APPENDIX 1

RESPIRATORY DISEASE MORTALITY TRENDS IN CANADA (1969-78)

Table A

Age-Standardized (all ages) Mortality Rates (Per 100,000) Canada 1969 - 1978

Year	Diseases of the Respiratory System (460-519)	Pneumonia (480-486)	Bronchitis, Emphysema and Asthma (490-493, 519.3*)	Other (460-474, 500-518, 519.0, 1, 2, 9)
Males				
1969	71.1	32.8	26.5	11.8
1970	70.6	31.0	27.3	12.3
1971	67.1	28.8	28.2	10.1
1972	70.1	29.6	27.8	12.7
1973	69.9	28.8	31.2	9.9
1974	70.2	29.5	30.6	10.1
1975	72.7	30.9	31.5	10.3
1976	70.6	27.4	32.2	11.0
1977	62.8	24.6	30.1	8.1
1978	65.2	24.2	32.0	9.0
% Change (1969-78)	-8.3%	-26.2%	+20.8%	-23.7%
Females				
1969	33.2	20.5	5.5	7.2
1970	32.8	18.9	6.4	7.5
1971	30.2	17.8	5.9	6.5
1972	30.8	17.8	5.9	7.1
1973	29.1	16.8	6.4	5.9
1974	28.2	16.5	6.5	5.2
1975	29.2	16.7	6.7	5.8
1976	29.6	15.3	7.2	7.1
1977	26.9	14.7	7.5	4.7
1978	25.2	12.7	7.6	4.9
% Change (1969-78)	-24.1%	-38.0%	+38.2%	-31.9%

* 519.3 - Statistics Canada code for Chronic Obstructive Lung Disease introduced in 1973.

Table B

Diseases of the Respiratory system, ICDA 460-519, Canada, 1969-1978

Year	Age-Specific Mortality Rates																		
	0-1	1-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
Males																			
1969	226.6	12.0	3.1	1.3	2.9	2.3	2.9	4.6	8.3	10.7	26.2	41.9	84.8	154.4	281.2	456.1	652.0	1095.2	2292.4
1970	220.0	11.3	2.3	2.3	2.6	2.7	3.7	3.4	7.3	12.3	23.4	46.0	82.5	156.0	271.6	476.0	686.3	1089.5	2128.4
1971	210.1	11.4	3.1	1.8	2.2	2.2	2.4	2.9	6.4	12.8	20.2	37.4	75.2	131.3	240.5	445.0	704.3	1084.0	2130.0
1972	200.8	9.2	2.3	2.0	2.5	3.4	3.0	4.4	5.0	10.8	21.2	42.9	66.1	134.8	272.1	444.8	745.4	1177.4	2284.6
1973	173.1	6.4	1.4	1.0	2.6	2.8	2.9	3.2	6.0	11.2	22.8	36.9	72.1	146.2	255.5	417.0	752.0	1234.2	2393.9
1974	143.6	8.4	1.2	1.7	2.8	2.9	2.4	3.8	6.5	11.2	20.4	35.6	72.2	135.7	259.0	436.1	711.2	1280.1	2530.8
1975	107.6	7.5	2.3	1.2	2.2	3.1	3.0	4.2	6.3	8.3	18.3	38.4	77.9	138.4	250.7	441.4	816.1	1327.4	2689.9
1976	99.6	9.4	2.0	1.5	2.5	2.5	2.9	2.4	4.5	8.6	18.4	32.4	72.3	144.1	244.6	444.9	771.3	1306.0	2597.0
1977	86.3	4.7	2.3	1.4	2.3	2.1	3.1	3.6	5.1	11.3	18.0	32.8	64.9	117.2	232.6	396.5	692.8	1217.2	2130.4
1978	75.4	5.6	1.3	1.8	1.3	2.0	2.5	2.6	3.5	11.8	17.1	32.3	61.1	130.0	244.6	425.8	699.1	1233.8	2359.5
Females																			
1969	173.5	10.5	1.8	1.7	3.2	2.0	2.8	4.7	6.0	9.9	17.7	22.8	34.8	44.1	87.7	142.4	255.9	495.9	1361.0
1970	173.9	11.4	2.8	2.3	2.8	2.5	3.6	4.9	6.9	8.4	14.1	22.1	34.5	49.7	77.7	146.4	257.0	522.3	1260.1
1971	168.7	12.1	1.7	2.2	2.3	2.9	2.3	4.5	5.5	7.2	11.7	17.6	33.4	41.5	67.3	123.1	233.4	469.2	1276.6
1972	144.8	9.3	2.0	1.1	2.2	2.2	2.4	3.9	6.0	9.5	12.9	19.7	32.6	47.3	76.5	142.1	266.0	467.5	1239.5
1973	110.4	8.5	1.4	1.9	1.7	2.0	2.5	4.1	6.2	7.9	9.0	24.0	26.7	46.4	78.6	123.7	241.5	501.2	1202.1
1974	98.2	8.1	1.4	1.3	1.1	2.4	1.9	3.4	4.5	7.4	12.1	19.3	27.7	48.2	74.5	112.1	240.3	462.3	1251.5
1975	94.1	7.6	1.6	1.3	1.6	1.9	2.0	2.6	4.2	7.7	11.3	18.6	29.3	48.6	85.2	140.3	262.1	478.5	1200.0
1976	71.1	6.4	1.4	1.6	1.0	1.3	1.5	2.5	4.9	7.0	12.2	15.2	33.6	41.7	79.5	138.2	253.9	527.7	1388.7
1977	61.7	4.2	1.3	1.6	1.9	1.0	1.9	2.6	4.4	7.7	11.6	17.2	29.5	45.0	73.0	129.7	242.1	444.8	1185.9
1978	56.5	4.0	1.6	0.5	0.9	1.8	2.1	2.6	3.5	6.4	8.5	14.1	27.4	43.6	80.0	127.4	213.8	434.6	1089.0

Note: Age-specific rates were calculated using population and death data obtained from Statistics Canada.

Source: Litven, W., Smith, H. and Wigle, D.T. Recent respiratory disease mortality trends in Canada. Chronic Diseases in Canada Vol. 1, No. 3, Dec. 1980.

APPENDIX 2

The Incidence and Severity of Pneumoconiosis in Canada (Adapted from Task Force on Occupational Respiratory Disease (Pneumoconiosis)

Agents		Abnormalities	Comments
1) Silica silicon dioxide, SiO_2)			
	crystalline: quartz tridymite cristobalite	silicosis	See Section 3.1.1
	amorphous: diatomite (kieselguhr, diatomaceous earth) flint	silicosis	" " "
2) Mixed dusts containing silica	kaolin	silicosis (kaolinosis)	" " "
	fuller's earth	silicosis	" " "
	aluminum and silica in bauxite smelting	Shaver's disease	Rapidly progressive disabling disease
	other mixed dust	mixed pneumoconiosis	
3) Other silicon compounds: silicon carbide (syn. carborundum, SiC)	pneumoconiosis		Etiological specificity not firmly established.
		due to ...	Disabling silicosis may occur from ingredients in manufacture of SiC.
Silicates	asbestos	asbestosis	See Section 3.1.1
	talc	talc pneumoconiosis	Progressive disease rarely leading to severe disability
	tremolite	talc pneumoconiosis	" " "
	actinolite	talc pneumoconiosis	" " "
	mica	mica pneumoconiosis	" " "
	aluminum silicates (e.g. sericite, muscovite group of sillimanite)	radiological abnormality	Probably only accumulation.
	glass fibres and particles	radiological abnormality	Probably only accumulation. Slight fibrosis? No disability.

Agents		Abnormalities	Comments
4) Coal and carbon	coal	coal workers' pneumoconiosis "black lung"	See Section 2.4
	graphite carbon black soot	graphite lung	Coal workers' pneumoconiosis possibly modified by content of silica varying widely according to source.
5) Aluminum		aluminosis	Rarely produces disabling disease
6) Iron Oxide		siderosis (welders' lung)	Only accumulation, possibly slight fibrosis, no disability
7) Hard Metals	sintered carbides of cobalt, tungsten, vanadium, etc.	hard metal pneumoconiosis	Progressive disease sometimes leading to severe disability
8) Tin		stannosis	Only accumulation of radio-opaque dust
9) Barium		baryosis	" " "
10) Beryllium		beryllium granulomatosis	Radiologically similar to other pneumoconioses but pathologically different. Severe disabling disease in which remission may occur.
11) Vanadium		vanadium lung	Radiologically resembles pneumoconiosis but clinically and pathologically is a pneumonitis
12) Manganese		manganese lung	" " "
13) Thomas' slag		Thomas' slag lung	" " "

APPENDIX 3

Table A

Labour Force by Selected Industries[1] in 1971 and Occupations at risk of Asbestos Exposure

	Canada			Provinces										
	Total	Males	Females	Nfld.	P.E.I.	N.S.	N.B.	Que.	Ont.	Man.	Sask.	Alta.	B.C.	Yukon N.W.T.
Mines	139.035	129,670	9,365	4,940	50	6,160	3,180	25,335	40,550	7,805	7,370	26,595	16,710	2,340
Metal mines	68,045	65,225	2,820	3,745	20	120	2,055	13,365	31,320	5,905	1,340	250	8,160	1,770
Placer gold mines	575	495	85	30	-	5	-	155	190	10	15	20	55	100
Gold quartz mines	5,790	5,620	175	-	-	-	-	1,265	3,900	20	5	15	160	440
Uranium mines	2,340	2,230	110	-	-	-	-	35	1,825	-	435	15	20	-
Iron mines	10,865	10,325	540	2,365	-	30	30	4,625	3,395	50	15	10	355	5
Miscellaneous metal mines	48,470	46,555	1,915	1,340	20	90	2,025	7,280	22,005	5,825	870	190	7,590	1,230
Mineral fuels	29,045	25,095	3,950	35	5	4,725	300	515	980	125	1,310	19,155	1,780	100
Coal mines	8,805	8,610	195	-	10	4,640	275	95	65	10	250	2,205	1,255	5
Crude petroleum and natural gas	20,240	16,485	3,755	35	-	90	20	425	920	115	1,065	16,950	530	95
Non-metal (except coal)	16,015	15,225	795	965	10	955	320	7,705	1,495	145	2,960	320	870	280
Asbestos	7,930	7,565	385	495	-	10	5	6,185	305	15	5	125	515	275
Peat extraction	1,470	1,390	80	5	10	20	300	700	50	65	40	55	235	-
Gypsum mines	690	650	40	100	-	485	5	20	30	15	5	10	30	-
Miscellaneous non-metal	5,925	5,635	295	370	-	435	15	810	1,115	55	2,905	125	90	5
Quarries and sand pits	6,770	6,480	290	25	10	180	135	1,965	2,935	670	110	350	605	-
Stone quarries	2,920	2,800	120	5	-	70	110	1,290	1,090	345	5	10	195	-
Sand pits or quarries	3,850	3,680	175	20	10	110	25	655	1,840	325	110	335	410	-
Services incidental to mining	19,155	17,650	1,505	165	5	180	370	1,800	3,810	1,170	1,655	6,515	3,295	190
Contract drilling for petroleum	4,680	4,380	295	35	-	40	10	90	290	35	550	3,455	150	10
Other contract drilling	3,300	3,140	155	80	-	40	55	750	1,110	505	165	55	415	55
Miscellaneous Services	11,175	10,125	1,050	65	-	95	305	960	2,405	545	935	3,055	2,730	125
Primary metal industries	117,060	109,760	7,275	180	20	3,580	775	26,565	70,910	2,330	1,130	3,430	7,940	-
Iron and steel mills	56,170	53,015	3,160	115	5	3,420	65	7,450	41,630	990	155	1,020	1,205	-
Steel pipe and tube mills	5,455	5,060	395	-	-	5	20	565	3,275	15	700	685	185	-
Iron foundries	10,990	10,415	580	15	10	110	55	2,200	7,150	310	55	425	580	5
Smelting and refining and aluminum rolling	36,485	34,200	2,285	45	-	25	625	14,105	13,900	860	210	1,250	5,490	-
Copper and copper alloy rolling, casting	3,530	3,250	285	-	-	5	-	1,070	2,140	10	-	10	300	-
Metal rolling, casting and extruding	4,405	3,830	570	10	-	20	15	1,165	2,820	165	10	35	185	-
Non-metallic mineral products industries	55,850	48,845	7,005	415	35	1,015	1,115	13,710	28,275	1,560	965	3,835	6,920	25
Clay product manufacturers	5,470	4,525	940	-	-	235	65	1,090	3,160	80	65	270	510	-
Cement manufacturers	5,445	5,100	345	135	5	115	130	2,020	1,510	195	130	395	820	-
Stone product manufacturers	1,270	1,130	140	15	5	75	60	515	390	50	40	55	30	20
Concrete products manufacturers	10,895	10,065	835	75	20	205	350	2,540	5,035	480	275	1,035	880	5
Ready mix product manufacturers	6,640	6,185	455	125	5	200	110	1,130	2,575	335	250	795	1,095	5
Glass and Glass Products	14,140	11,040	3,095	10	-	25	255	3,630	8,470	70	95	650	935	-
Abrasives	2,845	2,510	335	-	-	75	5	460	2,270	10	-	10	20	-
Lime manufacturers	735	705	30	5	-	5	35	115	385	85	-	80	25	-
Miscellaneous non-metallic mineral	8,415	7,590	825	40	-	85	110	2,205	4,480	250	90	565	605	-

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1) Source: 1971 Census of Canada, Catalogue 94-740, Volume III, Part 4, Statistics Canada, 1974.

Reproduced from Task Force on Occupational Respiratory Diseases (Pneumoconioses) Health and Welfare Canada, 1979.

Table B

Occupations at Risk for Asbestos Exposure in Mining, Milling, Manufacturing and Secondary Uses

Process	Products made or used	Jobs potentially at risk
1. Production Mining Milling		- rock mining, loading, trucking, crushing, milling
2. Handling		- transport workers, dockers, loaders, those who unpacked jute sacks (recently replaced with sacks which do not permit fibres to escape)
3. Primary uses in		
a) spray insulation	- spray of fibre mixed with oil	- spray insulators (construction, shipbuilding)
b) filler & grouting		
4. Manufacturing of		
a) textiles	- cloth, curtains, lagging, protective clothing, mailbags, padding, conveyor belts	- blending, carding, spinning, twisting, winding, braiding, weaving, slurry mixing, laminating, moulding, drying
b) cement products	- sheets, pipes, roofing shingles, gutters, ventilation shafts, flower pots	- blending, slurry preparation, rolling, pressing, pipe cutting
c) "paper" products	- millboard, roofing felt, fine quality electrical papers, flooring felt, fillers	
d) friction materials	- automotive products: gaskets, clutch plates, brake linings	
e) insulation products	- pipe and boiler insulation, bulkhead lining for ships	
5. Application		
a) construction		
new construction	- boards and tiles; putties, caulk & paints, joint fillers; cement products (tiles, pipes, siding, shingles); insulation materials	- directly: carpenters, laggers, painters, tile layers, insulation workers, sheet metal & heating equipment workers, masons; indirectly: all other workers on construction sites such as plumbers, welders, electricians
repair, demolition		
b) shipbuilding		
construction	- insulation materials (boards, mattresses, cloth) for engines, hull & decks, lagging of ventilation and water pipes, cables	- laggers, refitters, strippers, steam fitters, sailmakers, joiners, shipwrights, engine fitter masons, painters, welders, caulkers
repair, refits	- insulation materials, as described for construction	- directly: all above jobs on refits, dry dock and other repair operations indirectly: maintenance, fitters and repair men, electricians, plumbers, welders, carpenters
c) automotive industry		
manufacture	- gaskets, brake linings, undercoating	- installation of brake linings, gaskets, etc.
repair	idem	- service men, brake repairmen, auto mechanics

Reproduced from Task Force on Occupational Respiratory Diseases (Pneumoconiosis), Health and Welfare Canada, 1979.

APPENDIX 4

PNEUMOCONIOSIS ACROSS CANADA

Table A

New Cases Awarded Compensation, 1965 - 1976^a, 1980^b by Province

		1965	1966	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976	1980	Total
P.E.I.	Silicosis	-	-	-	-	-	-	-	-	-	-	-	-	N/A	2
N.B.	Silicosis	-	-	-	-	-	-	-	-	3	-	-	1	N/A	4
N.S.	C.W.P.	13	17	28	15	42	199	495	195	87	39	38	67	N/A	1,235
	Asbestosis	-	-	-	-	-	-	-	-	-	1	-	2	N/A	3
	Silicosis	16	18	9	26	26	41	35	29	47	36	39	19	57	341
Que.	Asbestosis	4	10	7	30	32	54	58	54	61	61	238	87	24	696
	Silicosis	-	2	-	-	1	3	-	1	6	-	-	-	10	13
Ont.	Silicosis	40	40	44	29	31	30	30	23	51	46	78	39	45	481^c
	Asbestosis	1	-	6	3	6	12	9	12	24	16	42	29	10	160
Man.	Silicosis	-	-	-	-	-	-	-	5	13	12	10	10	N/A	50
Sask.	Silicosis	-	-	-	-	-	-	-	-	1	1	-	1	-	3
	Asbestosis	-	-	-	-	-	-	-	-	1	1	1	-	-	3
Alta.	Silicosis	-	-	-	-	-	-	-	-	-	-	1	2	1	3
	Asbestosis	-	-	-	-	-	-	-	-	-	-	2	2	1	4
B.C.	Silicosis	14	17	15	24	10	7	9	14	7	11	12	11	17	151
	Asbestosis	-	-	-	1	1	-	1	2	-	2	1	3	11	11

a. Source: Provincial W.C.B. Questionnaire. Adapted from Task Force on Occupational Respiratory Diseases, (Pneumoconiosis), Health and Welfare Canada, 1979.

b. Source: Provincial W.C.B. Questionnaire sent out for this study.

c. 1965 - 1976 statistics on silicosis in Ontario are from the Ontario mining industry only. (Ontario statistics for silicosis in mines from 1977 to 1980 are 19,41,25 and 17 respectively.)

Table B

All Deaths Attributed to Pneumoconiosis[a] by Province, 1965-1974[b], 1979[c]

	Canada	Nfld.	P.E.I.	N.S.	N.B.	Que.	Ont.	Man.	Sask.	Alta.	B.C.	Yukon	N.W.T.
1965	95	-	-	8	1	20	40	3	1	3	19	-	-
1966	77		-	8	2	22	26	2	1	1	15	-	-
1967	89	-	-	8	1	19	37	2	1	3	18	-	-
1968	106	1	-	9	3	33	32	3	1	3	21	-	-
1969	73	1	-	6	-	16	32	-	-	3	15	-	-
1970	75	1	-	3	3	21	26	1	-	6	13	1	-
1971	79	-	-	11	-	15	33	1	2	2	14	1	-
1972	80	1	-	11	2	18	27	1	1	2	17	-	-
1973	64	1	-	7	2	18	25	-	-	2	9	-	-
1974	75	-	-	10	1	20	29	1	-	1	13	-	-
1979	67	-	-	6	4	12	22	3	-	3	17	-	-

a ICDA Codes: 1965-68: 001 and 523 and 524; 1969-74: 010 and 515 and 516.0
1979: 500-505 and 507.1.

b. Source: Statistics Canada: Causes of Death, 1965-74. Adapted from Task Force on Respiratory Disease (Pneumoconiosis), Health and Welfare Canada, 1979.

c. Source: Statistics Canada: Causes of Death, 1979, Catalogue 84-203.

APPENDIX 5

INCIDENCE OF RADIOLOGICAL SILICOSIS

Table A

New 5 Ratings By Period So Rated[a] (excluding original examinations)

Period Rated New 5	Ontario Exposure Only				Exposure Outside Ontario				Total			
	Uranium Only	Uranium And Other Metal(s)	Other than Uranium	Total	Uranium Only	Uranium And Other Metal(s)	Other than Uranium	Total	Uranium Only	Uranium And Other Metal(s)	Other than Uranium	Total
Before 1930	-	-	18	18	-	-	8	8	-	-	26	26
1930 - 1934	-	-	120	120	-	-	88	88	-	-	208	208
1935 - 1939	-	-	146	146	-	-	92	92	-	-	238	238
1940 - 1944	-	-	164	164	-	-	96	96	-	-	260	260
1945 - 1949	-	-	153	153	-	-	78	78	-	-	231	231
1950 - 1954	-	-	89	89	-	-	64	64	-	-	153	153
1955 - 1959	-	1	76	77	-	1	64	65	-	2	140	142
1960 - 1964	-	1	57	56	3	7	49	59	3	8	106	117
1965 - 1969	1	12	67	80	10	27	44	81	11	39	111	161
1970 - 1974	5	24	42	71	21	38	25	84	26	62	67	155
1975	3	20	22	45	11	25	10	46	14	45	32	91
1976	2	11	24	37	3	10	8	21	5	21	32	58
1977	3	5	11	19	3	8	6	17	6	13	17	36
1975 - 1977	8	36	57	101	17	43	24	84	25	79	81	185
Total	**14**	**74**	**989**	**1,077**	**51**	**116**	**632**	**799**	**65**	**190**	**1,621**	**1,876**

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a. Source: Wheeler, W.C. Statistics on Silicosis Among Miners in Ontario 1930 - 1977. Statistical Analysis Section, Ontario Workers' Compensation Board.

Table B

New 4 Ratings[a] By Period So Rated[b]
(excluding original examinations)

	Ontario Exposure Only				Exposure Outside Ontario				Total			
Period Rated New 5	Uranium Only	Uranium And Other Metal(s)	Other than Uranium	Total	Uranium Only	Uranium And Other Metal(s)	Other than Uranium	Total	Uranium Only	Uranium And Other Metal(s)	Other than Uranium	Total
Before 1930	-	-	-30	30	-	-	9	9	-	-	39	39
1930 - 1934	-	-	203	203	-	-	145	145	-	-	348	348
1935 - 1939	-	-	335	335	-	-	204	204	-	-	539	539
1940 - 1944	-	-	424	424	-	-	233	233	-	-	657	657
1945 - 1949	-	-	265	265	-	-	157	157	-	-	422	422
1950 - 1954	-	-	168	168	-	-	122	122	-	-	290	290
1955 - 1959	-	11	263	274	-	13	168	181	-	24	431	455
1960 - 1964	-	20	190	210	10	30	99	139	10	50	289	349
1965 - 1969	3	30	211	244	19	57	110	186	22	87	321	430
1970 - 1974	11	56	126	193	35	69	57	161	46	125	183	354
1975	3	11	25	39	1	10	1	12	4	21	26	51
1976	4	10	17	31	2	7	14	23	6	17	31	54
1977	-	4	4	8	2	10	5	17	2	14	9	25
1975 - 1977	7	25	46	78	5	27	20	52	12	52	66	130
Total	21	142	2,261	2,424	69	196	1,324	1,589	90	338	3,585	4,013

a - "Code 4" is considered "Dust effects" only. See text for explanation.
b - Source: Ibid.

APPENDIX 5

Table C
New 5 Ratings By Period So Rated[a]
(excluding original examinations)

	Ontario Exposure Only				Exposure Outside Ontario				Total			
Period Rated New 5	Gold Only	Gold And Other Metal(s)	Other than Gold	Total	Gold Only	Gold And Other Metal(s)	Other than Gold	Total	Gold Only	Gold And Other Metal(s)	Other than Gold	Total
Before 1930	4	12	2	18	2	4	2	8	6	16	4	26
1930 - 1934	36	66	18	120	31	42	15	88	67	108	33	208
1935 - 1939	73	63	9	145	54	34	3	91	127	97	12	236
1940 - 1944	94	62	9	165	52	41	1	94	146	103	10	259
1945 - 1949	94	55	4	153	43	33	2	78	137	88	6	231
1950 - 1954	50	36	2	88	29	30	4	63	79	66	6	151
1955 - 1959	57	19	1	77	27	31	7	65	84	50	8	142
1960 - 1964	45	9	4	58	29	22	8	59	74	31	12	117
1965 - 1969	45	28	7	80	24	34	23	81	69	62	30	161
1970 - 1974	22	28	14	64	11	30	37	78	33	58	51	142
Total	**520**	**378**	**70**	**968**	**302**	**301**	**102**	**705**	**822**	**679**	**172**	**1,673**

[a] - Source: Wheeler, W.C. Statistics on Silicosis Among Gold Miners in Ontario 1930 - 1974, Statistical Analysis Section, Ontario Workers Compensation Board, Sept. 1976.

APPENDIX 5

Table D

New 4 Ratings[a] By Period So Rated (excluding original examinations)

	Ontario Exposure Only				Exposure Outside Ontario				Total			
Period Rated New 5	Gold Only	Gold And Other Metal(s)	Other than Gold	Total	Gold Only	Gold And Other Metal(s)	Other than Gold	Total	Gold Only	Gold And Other Metal(s)	Other than Gold	Total
Before 1930	3	13	14	30	-	2	7	9	3	15	21	39
1930 - 1934	79	83	41	203	73	56	16	145	152	139	57	348
1935 - 1939	174	117	43	334	109	79	14	202	283	196	57	536
1940 - 1944	259	125	40	424	138	85	10	233	397	210	50	657
1945 - 1949	173	82	10	265	101	50	5	156	274	132	15	421
1950 - 1954	96	64	7	167	57	60	5	122	153	124	12	289
1955 - 1959	154	96	23	273	89	72	19	180	243	168	42	453
1960 - 1964	98	64	44	206	52	53	33	138	150	117	77	344
1965 - 1969	104	61	76	241	31	77	67	175	135	138	143	416
1970 - 1974	56	70	47	173	16	54	65	135	72	124	112	308
Total	**1,196**	**775**	**345**	**2,316**	**666**	**588**	**241**	**1,495**	**1,862**	**1,363**	**586**	**3,811**

a - "Code 4" represents "Dust effects". See text for explanation. This Table excludes 4 ratings with less than 60 months total dust exposure and 4 ratings with no dust exposure in Ontario.

APPENDIX 6

Chronic Obstructive Lung Disease (Ottawa and Sudbury)

Table A

Mean FEV_1 FVC ratios* for samples of residents of Sudbury (S) and Ottawa (O), by sex, age and smoking habits.

Age (yr) \ No. of cigarettes smoked /d		Males					Females					Both sexes	
		0	1-9	10-24	25+	Mean	0	1-9	10-24	25+	Mean	Mean	
14-24	S	0.866	0.824	0.859	0.845	0.849	0.877	0.872	0.857	0.772	0.845	0.847	0.856
	O	0.888	0.869	0.875	0.795	0.857	0.886	0.870	0.842	0.885	0.871	0.864	
25-44	S	0.833	0.783	0.742	0.757	0.780	0.818	0.772	0.794	0.800	0.796	0.788	0.809
	O	0.870	0.848	0.795	0.805	0.831	0.848	0.820	0.838	0.796	0.826	0.829	
45-64	S	0.815	0.726	0.741	0.722	0.751	0.804	0.755	0.783	0.792	0.784	0.768	0.792
	O	0.829	0.774	0.781	0.781	0.791	0.848	0.830	0.832	0.840	0.838	0.815	
All ages	S	0.838	0.778	0.783	0.775	*0.793*	0.833	0.800	0.811	0.788	*0.806*	*0.801*	
	O	0.862	0.830	0.818	0.794	*0.826*	0.861	0.840	0.837	0.840	*0.845*	*0.836*	
Both cities		0.850	0.804	0.801	0.785	*0.810*	0.847	0.820	0.824	0.814	*0.826*	*0.818*	

Table B

Prevalence* of chronic bronchitis (per 1,000) for samples of residents of Sudbury (S) and Ottawa (O), by sex, age and smoking habits.

Age (yr) \ No. of cigarettes smoked /d		Males					Females					Both sexes	
		0	1-9	10-24	25+	Mean	0	1-9	10-24	25+	Mean	Mean	
14-24	S	49	49	47	118	63	15	27	68	91	49	56	50
	O	4	0	37	52	23	16	54	87	91	62	43	
25-44	S	62	103	43	170	95	33	87	86	130	84	90	90
	O	10	49	83	245	97	20	32	70	209	83	90	
45-64	S	59	222	247	179	177	27	74	102	238	110	144	120
	O	13	0	133	235	95	35	43	86	229	99	97	
All ages	S	57	125	112	156	*112*	25	61	85	153	*81*	*97*	
	O	9	16	84	177	*72*	25	43	81	176	*81*	*77*	
Both cities		33	71	99	167	*92*	25	52	83	165	*81*	*87*	

*Italicized figures represent overall mean ratios, with age and smoking habits considered simultaneously.

Source: Reproduced from Neri, L.C., Mandel, J.S., Hewitt, D. and Jurkowski, D. C.M.A.J. 113: 1045, 1975.

APPENDIX 7

CHRONIC OBSTRUCTIVE LUNG DISEASE - CONVERTER PLANT

Table A

Number of Men by Exposure Group and Smoking Status With and Without Symptoms of Chronic Bronchitis

		Refinery		Converter	
		No.	**%**	**No.**	**%**
Chronic Bronchitis	Never Smoked	1	11.1	1	2.44
	Former Smokers	1	4.35	7	8.75
	Smokers	3	9.68	62	32.80
	Total	**5**	**7.81**	**70**	**22.58**
No Bronchitis	Never Smoked	8	88.89	40	97.56
	Former Smokers	22	95.65	73	91.25
	Smokers	29	90.62	127	67.20
	Total	**59**	**92.19**	**240**	**77.42**
	Grand Total	**64**	**100.00**	**310**	**100.00**

Source: Chronic Obstructive Lung Disease Among Persons Employed for Ten Years or More in the converter plant at Copper Cliff, Ontario. Aug./Sept. 1973. Ministry of Health, March 1976.

Table B

Mean Values of FVC and FEV$_1$ with Percentage of Predicted Values in Refinery and Converter Groups

	F V C				F E V$_1$				F E V$_1$/ F V C %			
	MEAN	% OF PREDICTED VALUE			MEAN	% OF PREDICTED VALUE			MEAN OF INDIV. RATIOS			No.
Refinery	4302cc.	101.67 SD ± 12.61	Never Smoked	103.33 SD ± 16.20	3294cc.	99.41 SD ± 13.83	Never Smoked	101.00 SD ± 17.20	76.78	Never Smoked	78.67 SD ± 4.12	9
			Former Smokers	104.39 SD ± 12.65			Former Smokers	102.9 SD ± 11.61		Former Smokers	77.35 SD ± 6.50	23
			Smokers	99.81 SD ± 11.52			Smokers	96.44 SD ± 14.06		Smokers	75.84 SD ± 7.01	32
Difference Never Smoked Current Smokers				3.52 $p > 0.05$				5.56 $p > 0.05$			2.83 $p > 0.95$	
Converter	4070cc.	96.16 SD ± 14.04	Never Smoked	100.00 SD ± 12.88	3093cc.	91.67 SD ± 18.74	Never Smoked	101.00 SD ± 15.50	74.75	Never Smoked	79.29 SD ± 5.74	41
			Former Smokers	99.98 SD ± 12.63			Former Smokers	97.00 SD ± 16.14		Former Smokers	76.41 SD ± 8.98	80
			Smokers	93.71 SD ± 14.36			Smokers	87.35 SD ± 19.27		Smokers	73.05 SD ± 10.20	189
Difference Never Smoked Current Smokers				6.29 $p < 0.01$				13.65 $p < 0.001$			6.24 $p < 0.001$	
Difference and Standard Error	232cc SE ± 91cc ($p=0.011$)	5.51 SE ± 1.77			201cc SE ± 84cc ($p=0.017$)	7.74 SE ± 2.03			2.03 SE ± 0.98 ($p=0.038$)			

Source: Ibid.

Table C

FEV_1/FVC Ratio, Graded for Severity, by Occupational Group and Smoking Status

	FEV_1/FVC %	Normal (70%)		Slight (60-69%) Obstruction		Moderate to Severe (59%) Obstruction		Total
		No.	%	No.	%	No.	%	No.
Refinery	Never Smoked	9	100.0	-	-	-	-	9
	Former Smokers	20	87.0	3	13.0	-	-	23
	Smokers	26	81.2	6	18.8	-	-	32
	Total	**55**	**85.9**	**9**	**14.1**	-	-	**64**
Converter	Never Smoked	40	97.6	1	2.4	-	-	41
	Former Smokers	68	85.0	6	7.5	6	7.5	80
	Smokers	135	71.4	34	18.0	20	10.6	180
	Total	**243**	**78.4**	**41**	**13.2**	**26**	**8.4**	**310**

Source: Ibid.

APPENDIX 8

CHRONIC BRONCHITIS AND PULMONARY IMPAIRMENT IN ELLIOT LAKE MINING OPERATIONS

Table A

Prevalence of Bronchitis by Age and Smoking Habits[a]

Age	Never Smoked		Former Smokers		Smokers		Total
- 24	0/21[a]	(0.0%)	0/21	(0.0%)	4/67	(6.0%)	3.7%
25-34	1/32	(3.1%)	2/33	(6.1%)	15/174	(8.6%)	7.5%
35-44	3/47	(6.4%)	1/73	(1.4%)	32/172	(18.6%)	12.3%
45-54	2/33	(6.1%)	5/61	(8.2%)	33/135	(24.4%)	17.5%
55+	2/10	(20.0%)	9/49	(18.4%)	9/46	(19.6%)	19.0%
Total	**5.6%**		**7.2%**		**15.7%**		**12.1%**

Notes

[a] Source: Survey of Certain Conditions of the Respiratory Organs Among Persons Employed Underground and in Surface Crushers and Mills of Two Operating Mines at Elliot Lake, Ontario. Feb. 17-March 30, 1974, Ministry of Health, Ontario. April 1975.

The figure above the oblique line refers to those with chronic bronchitis and that below the line the number of individuals in that particular group.

Table B

Multiple Regression Analysis
Using Only Smokers and Non-Smokers

Dependent Variable	Independent Variables							
	Effect of Age		Effect of Height		Effect of "Dust Exposure"		Effect of Smoking	
	A	t Ratio	H	t ratio	E	t ratio	S	t ratio
FVC	-0.0201	6.72+	0.1707	18.06+	-0.0387	14.43+	-0.0770	1.33^{nss}
FEV_1	-0.0239	8.73+	0.1248	14.47+	-0.0418	17.11+	-0.1985	3.82+
MMFR	-0.0324	5.83+	0.0880	5.02+	-0.0602	12.13+	-0.5616	5.33+
Bronchitis	-0.0005	0.29^{nss}	-0.0054	1.02^{nss}	0.0054	3.59+	0.1078	3.40+
Breathlessness	0.0048	2.24*	-0.0046	0.68^{nss}	0.0090	4.70+	0.0540	1.33^{nss}
Wheezing	-0.0023	1.59^{nss}	-0.0059	1.28^{nss}	0.0016	1.25^{nss}	0.0932	3.35+

nss = Non Significant

* = Significant P 0.05

+ = Significant P 0.001

Number = 737 (smokers and non-smokers)

A; E; S; H = regression co-efficient

Source: Ibid.

Table C

Prevalence of Chronic Bronchitis in 3 Populations in Ontario

	Age Group	% Chronic Bronchitis
Ottawa Males	25 - 64	8.1%
Sudbury Males	25 - 64	13.6%
Elliot Lake Miners	25 - 64	13.1%
Elliot Lake Uranium Workers (all)	average 39	12.1%
Ottawa Males	45 - 64	9.5%
Sudbury Males	45 - 64	17.7%
Elliot Lake Uranium Workers (24 or more years of service)	average 54	16.4%

Source: Ibid.

APPENDIX 9

COMPARISON OF PULMONARY IMPAIRMENT RATING SCHEMES

Table A

Classification of Impairment

	Class I	Class II		Class III		Class IV		Class V	
Dyspnea	(No impairment The subject may or may not have dyspnea. If dyspnea is present it is for non-respiratory reasons.	(Mild Impairment). Dyspnea on fast walking on level ground or walking up a hill.		(moderate Impairment) Dyspnea walking on level ground with person of the same age or walking one flight of stair.		(Severe Impairment) Dyspnea after walking more than 100 meters at own pace on leve ground.		(Very Severe Impairment) Dyspnea on talking or undressing or unable to leave his dwelling because of breathlessness.	
Pulmonary Function Tests		Obstructive	Restrictive	Obstructive	Restrictive	Obstructive	Restrictive	Obstructive	Restrictive
FVC	Normal[+]	N/A	More than 60% of Predicted Value	N/A	40%-60% of Predicted Value	N/A	Less than 40% of Predicted Value	Same as IV	Same as IV
FEV$_1$	Normal	More than 60% of Predicted Value	N/A	40%-60% of Predicted Value	N/A	Less than 40% of Predicted Value	N/A	Same as IV	Same as IV
DLCO	Normal	Normal or slightly abnormal	Less than 75% of Predicted Value	Usually more than 50% of Predicted Value	Usually more than 50% of Predicted Value	Reduced	Less than 40% of Predicted Value	Same as IV	Same as IV
Maximal VO$_2$	Normal more than 25ml/Kgm/min[*]	Less than 25 ml/Kg/min[*]	Less than 25 ml/Kg/min	Less than 15 ml/Kg/min[*]	Less than 15 ml/Kg/min[*]	Less than 7 ml/Kg/min	Less than 7 ml/Kg/min	Less than 7 ml/Kg/min	Less than 7 ml/Kg/min
Arterial blood gases at rest	Normal	Normal	Normal	PO$_2$ more than 60 mmHg PCO$_2$ less than 45 mmHg	PO$_2$ more than 60 mmHg PCO$_2$ less than 45 mmHg	PO^2 more than 60 mmHg PCO$_2$ less than mmHg	PO$_2$ more than 60 mmHg PCO$_2$ less than 45 mmHg	PO$_2$ less than 50 mmHg PCO$_2$ less than 45 mmHg	PO$_2$ less than 50 mmHg PCO$_2$ variable

[+] A normal value lies within 2 standard deviations of the mean. The mean value is standardized for age, sex and height when applicable.

[*] If less, this limitation is secondary to non-pulmonary reasons.

Source: Task Force on Occupational Respiratory Diseases (Pneumoconiosis), Health and Welfare Canada, 1979.

Table B

Schemes for Definition of Severe Pulmonary Impairment[a]

Reference	Definition of Severe Impairment	Obstructive Criteria	Interstitial Criteria	Remarks
Epler et al[a]	Classes III & IV[h]	FEV_1/FVC 40% or FEV_1 40% pred.[l]	FVC 50% pred. Dsb 40% pred.	Predicted values of Kory et al.[l]
Social Security[b]	Total and permanent disability	MVV and FEV_1	FVC Dsb 30% pred. PO_2-PCO_2 criteria	Height limits[j]
Veterans Administration[c]	60-100% impaired	Dyspnea grade 3 or 4	Dyspnea grade 3 or 4	--
American Medical Association[d]	Class IV 60-90% impaired	FVC, FEV_1, MVV_F 55% pred. (2 of 3)	FVC, FEV_1, MVV_F 55% pred.[l] (2 of 3)	Predicted values of Kory, et al.[l]
Wilson et al.[e]	Classes IV and V	DPD 250[k],	DPD 250[k]	DPD = Degree of Pulmonary Disability[k]
Coal Miner's Act[f]	Total and permanent disability	FEV_1 and MVV_F 60 % pred.	FEV_1 and MVV_F 60 % pred.	FEV_1 predicted values from Knudson et al.[l] (MVV = FEV_1 X 40)
Canadian Task Force[g]	Class IV and V	FEV_1 40% pred.[l]	FVC 40% pred. DL_{CO} 40% pred. PO_2-PCO_2 criteria VO_2 max. criteria	

Definition of abbreviations:

FEV_1, FVC, PO_2-PCO_2, VO_2 max--see text; % pred. = % of the predicted normal value; MVV = maximum voluntary ventilation; D_{sb} = single breath diffusing capacity, DL_{CO} = diffusing capacity for carbon monoxide (the latter two measure the same parameter and are used synonomously when the latter is done by the "single breath" method.).

Notes

a. This table was largely adapted from: Epler, G.R., Saber, F.A. and Gaensler, E.A. Determination of severe impairment (disability) in interstitial lung disease Am. Rev. Res. Dis. 121: 647, 1980.

b. U.S. Dept. of HEW, Social Security Administration. Disability Evaluation Under Social Security: A Handbook for Physicians DHEW Publ. No. 79-10089; 23, 1979. Cited by Epler, 1980, op. cit.

c. Veterans Administration. Schedule for Rating Disability: Section 6600 for Bronchitis, p. 72-3R; 6603 for Emphysema, p. 75-2R; 6802 for Pneumoconiosis, p. 78-3R, Sept. 1975, cited by Epler, 1980, op. cit.

d. Committee on Rating of Mental and Physical Impairment. The respiratory system J.A.M.A. 194: 177, 1965.

e. Wilson, R.H., Hargis, B.J., Honr, R.L. and Shields, D.O. A clinical and laboratory method of determining degree of pulmonary disability with a proposed classification. Am. J. Med. 37: 251, 1964, cited by Epler, 1980, op. cit.

f. Dept. of Labour, Employment Standards, Administration. Standards for Determining Coal Miner's Total Disability or Death Due to Pneumoconiosis, Fed. Reg. Part IV: p.p. 17722-3, Apr. 1978, cited by Epler, 1980, op. cit.

g. Task Force on Occupational Respiratory Disease (Pneumoconiosis), Health and Welfare Canada, Feb. 1979.

h. See Table A in this Appendix for definition.

i. Kory, R.C., Callahan, R., Boren, H.G., et al. The veterans administration - army cooperative study of pulmonary function I. clinical spirometry in normal man. Am. J. Med. 30: 243, 1961.

j. Limits were based on height without consideration of age or sex; e.g. the upper limit for 68 inches tall are MVV_F: 43 L/min; FEV_1: 1.3 L; and FVC: 1.8 L. a 45 year-old man, this corresponds to 40% predicted values for that individual.

k. DPD for impaired gas exchange: $DPD = 113.85 + 0.818\ MVV_F + 6.60\ Dsb + 44.04\ FEV_1 - 1.818\ age$.

DPD for obstructive disease: $DPD = 213.6 + 1.88\ MVV_F = 74.5\ FEV_1 - 1.76\ age - 34.62\ FVC$.

l. Knudsen, R.J., Slatin, R.C., Lebowitz, M.D. and Burrow, B. The maximum expiratory flow-volume curve. Am. Rev. Respir. Dis. 113: 587, 1976.

APPENDIX 10

Variability in Interpretation of Pulmonary Function Tests

Table A

Distribution of Responses Categorizing Pulmonary Function Test Results[1]

	University Group	Independent Physicians
Obstructive Disease:		
Total Compared	780	780
Agreement	57 %	60 %
Differed by 1 Category	41 %	37 %
Differed by More Than 1 Category	2 %	3 %
Restrictive Disease		
Total Compared	390	390
Agreement	63 %	65 %
Differed by 1 Category	32 %	32 %
Differed by More Than 1 Category	5 %	3 %

Note

a. Source: Cory, J., Huseby, J., Culver, B. and Kosanke, C. Variability in Interpretation of Pulmonary Function Tests. Chest 76: 389, 1979.

Table B

Variation in Obstructive Disease Categories Using FEV_1/VC Ratios[a]

	Normal (%)	Mild (%)	Moderate (%)	Severe (%)
Gaensler and Wright '66	75	76-60	59-40	40
ACCP '67	80	80-65	64-50	50
Ellis '65	80	80-67	66-52	52
Utah '75	70	69-62	61-44	44
Agreement	80	69-67	59-52	40
Disagreement		80-70	66-60	51-40

a. Source: Ibid. (References cited by Cory et al.)

B. THE COMPENSATION OF OCCUPATIONAL RESPIRATORY DISEASES

1. The Facts, Figures and Issues Involved

The problems of compensating respiratory disease are twofold. First, the causation issue must be sorted out; a decision has to be made regarding the work-relatedness of a given disease and the affected workers must be identified and reported to the WCB. Secondly, an assessment must be made of the disability attributable to the condition in question, more often than not in the presence of other, perhaps non-work-related conditions; a decision also has to be made regarding the advisability or ability of the individual to continue working with this disability.

Both work-relatedness of specific conditions and level of disability have been the focus of controversy. If the White Paper recommendations for a wage loss scheme were to be enacted, the issue of 'fitness for work' would be of even greater significance.

1.1 Identifying Work-Related Diseases and the Work-Relatedness of Disease

For the non-specific or multifactorial diseases (for example, the chronic obstructive lung diseases), the decision of work-relatedness is the paramount issue. For those workers who develop well-recognized occupational diseases (e.g. pneumoconiosis), concerns revolve less around proving their work-relatedness than around the level of disability attributable to the condition. However, an issue which involves the "occupationally-specific" diseases as well as the non-specific potentially work-related diseases, is the identification of workers who have become victims of workplace hazards so that they may receive the compensation to which they are entitled.

Several respiratory conditions have been recognized in this province as work-related. These include silicosis, asbestosis, other pneumoconioses, occupational asthma, and cases of chronic obstructive lung disease and emphysema where guidelines specifically apply. The evolution of policy and specific issues with respect to the adjudication of each of these diseases will be discussed in subsequent sections.

1.1.1 Identification of Workers with Work-related Respiratory Diseases

Steps have been taken to monitor for diseases in plants where recognized hazards are known to exist. As shown in Table IIIB-1 in the fiscal year ending March 1981, over 8 thousand workers were given chest x-rays by the Occupational Health Branch of the Ministry of Labour as a result of their exposure to silica dust; over 3 thousand asbestos-exposed workers were x-rayed; about 1 thousand isocyanate-exposed workers and 4 thousand workers exposed to other hazardous dust also received chest x-rays through government monitoring programs. Respiratory function tests were performed by the Ministry on some 17,000 workers in 684 companies. In this series of government monitoring, radiological evidence of silicosis was reported in 84 workers, 18 of whom were newly diagnosed, and 27 cases of asbestosis were identified, including 8 newly diagnosed. The number of cases of respiratory disease discovered as a result of the pulmonary function testing was not specified.

Table IIIB-1

Ministry of Labour Respiratory Disease Monitoring Program
March 1980 - March 1981[a]

Chest X-Rays

	No. of Workers	No. of Companies Involved	Exposure	Newly Diagnosed Disease
	8,427		Silica Dust	18 - Silicosis
	3,781		Asbestos	27 - Asbestosis
	1,052		Isocyanates	N/A[c]
	4,174		Other Hazardous Dusts	N/A[c]
Total	17,434			
Pulmonary Function Tests				
	11,825	510	Inorganic Dusts	N/A[c]
	1,166	54	Isocyanates	N/A[c]
	470	9	Organic Dusts	N/A[c]
	3,576	111 [b]	Silica Dust	N/A[c]
Total	17,037			

Notes

a. Source: 1981 Ministry of Labour Annual Report.

b. Foundries only included.

c. N/A - data not available.

Statistics on the origin of most Workers' Compensation Board claims are not generally kept. The sample survey conducted for this Inquiry identified "government doctor" as initiator of the claim in only 2.7% of the 150 "silicosis" claims reviewed. Other physicians filed 18.7% of claims from this index; 9.3% originated from the employer, 37.3% from the claimant, 12.7% from the union and in 19.3% no single person or group could be identified. In a similar study carried out by Barth for the Dupre Commission, 41% of claims were set up by the worker or his survivor, 31% by the union, 10% by employers and 18% by the health community. (See Table IIIB-2.)

These data, can be interpreted in a number of ways. Although it is recognized that the person or group to file the first piece of mail may not truly be the initiator of the claim, the fact that the claimant or union initiated 62-72% of the claims may indicate an inadequacy in the "official" identification procedure. It should be noted that only current employees are included in the medical monitoring programs and it is not known how many of the claimants were retired or even deceased. Moreover, as will be shown below, many of the "silicosis" claims, for example, were filed on the basis of the worker having a respiratory disease despite the lack of x-ray evidence of silicosis. This reveals a definite divergence of opinion as to what constitutes an occupational disease in workers exposed to dusty conditions. While the data presented in Table IIIB-3 indicate that it is mainly those workers with x-ray evidence of silicosis (or other pneumoconiosis) who receive compensation for chronic respiratory disease, many workers seem to think that other respiratory symptoms which they experience are indeed work-related.

Thus, monitoring and reporting systems depend not only on the extensiveness of the system but on the definition of what is being sought by the program, i.e. what is defined as work-related. The fact that there was a significant difference in the outcome of the claims initiated by health professionals and employers as compared to those initiated by workers and their representatives is also of interest in this regard. (See Tables IIIB-4a and IIIB-4b.)

As shown in Table IIIB-3, in 1980 there were 45 workers who received compensation for silicosis, 10 for asbestosis, 8 for other pneumoconiosis and 49 workers with respiratory conditions due to chemical fumes and vapours; 28 workers received compensation for bronchitis, emphysema and asthma (including 21 for asthma, 5 for chronic bronchitis and two unspecified), and four workers received compensation for other non-malignant lung conditions (excluding infections). Thus the total number of workers defined to have a work-related occupational respiratory disease was 144.

Of immediate note is that a maximum of only 28 workers with chronic obstructive lung disease (some of the asthma cases may have been acute only) received compensation. However, chronic obstructive lung disease accounts for considerable mortality in Ontario, at least 100 times this figure, not to mention the illness, suffering and hospitalization (over 5,000 times this number)

Table IIIB-2

Silicosis and Asbestosis Claims by Person/Group Who Filed First Piece of Mail

Person/Group Who Filed Claim	"Silicosis" Sample Survey[a] Claims	Percent	Asbestos-Related Claims[b] Percent
Employer 14	9.3%	10%	
Attending Physician[c]	28	18.7%	18%
Gov't Physician[c]	4	2.7%	-
Union 19	12.7%	31%	
Claimant[d] 56	37.3%	41%	
Other[e] 29	19.3%	-	
Total 150	**100.0%**	**100%**	

Notes

a. This sample included only claims registered in the silicosis index maintained by the Program Planning and Statistical Services Branch of the WCB. This survey, carried out in the fall of 1981 specifically for this Inquiry, reviewed 150 silicosis claims set up prior to July 1, 1979.

b. This sample included 156 asbestos-related claims of which 101 were for asbestosis only. This review was carried out by Dr. Peter Barth for the Royal Commission on Asbestos and included claims set up 1976-1980.

c. Whereas the silicosis review attempted to separate attending physician, company physician and Government physician, Barth considered the Health Community as one group.

d. Barth grouped "worker or his survivor" in one category.

e. "Other" in the silicosis survey represented claims where no single person/group could be identified by the coders.

Table IIIB-3

Allowed Compensation Claims for Non-Malignant Respiratory Disease, 1980[a]

Pneumoconioses - Total			112
Silicosis		45	
Asbestosis		10	
Other Pneumoconioses		57	
Pneumopathy due to inhalation of other dust including extrinsic allergic alveolitis, i.e. farmers' lung, bagassosis, byssinosis grain handlers' disease	8		
Respiratory conditions due to chemical fumes and vapours (inhalation) (usually acute only)	49		
Bronchitis, Asthma and Emphysema - Total			28
Bronchitis (not specified as acute or chronic)		2	
Chronic Bronchitis		5	
Asthma		21	
Other diseases of lung (non-malignant, non-infective)			4
Total allowed (non-maligant, non-infective) respiratory disease claims =			144

Note

a. Source: Occupational Diseases - 1980 First Payment Compensation Claims, Program Planning and Statistical Services Branch, July 20, 1981.

Table IIIB-4a

Count of Claims Received for Respiratory Conditions by First Claim Status and Person/Group Who Filed First Piece of Mail[a]

Person/ Group Who Filed Claim	First Claim Status Allowed Medical Aid Only	Allowed Compensation and Medical Aid	Rejected Claims	Claimant Did Not Reply	Total Claims
Employer	1	7	4	2	14
Claimant	5	11	35	5	56
Union	1	1	15	2	19
Family Doctor	2	11	12	3	28
Company Doctor	-	-	-	-	-
Gov't Doctor	-	1	3	-	4
Other[b]	2	9	17	1	29
Total	**11**	**40**	**86**	**13**	**150**

Notes

a. This sample study includes only "silicosis" claims set up prior to July 1, 1979.
b. "Other" represents claims where no single Person/Group could be identified: 37.9% of the "Other" claims were allowed.

Table IIIB-4b

Outcome of Silicosis Claims Initiated by Employers or Health Professionals Compared to those Initiated by Claimant or Union

	Allowed		Rejected or No Reply		Total	
	Claims	%	Claims	%	Claims	%
Health Professionals & Employers	22	47.8%	24	52.2%	46	100.0%
Claimant & Union	18	24.0%	57	76.0%	75	100.0%
Total	**40**	**33.1%**	**81**	**66.9%**	**121**	**100.0%**

X^2 = 7.79, 01; P $\overset{>}{=}$ 0.01 highly significant attributable to these respiratory conditions (see Section 2.1 in Part A). As shown by the many studies discussed in Section 3 in the first part of this chapter, although cigarette smoking is clearly the predominant cause of chronic obstructive lung disease, occupational factors at least contribute to the morbidity. The surveys referred to in Part A revealed that for every case of compensated respiratory illness, there were at least five cases of respiratory illnesses that were not compensated, but believed to have been related at least in part to workplace factors.

According to the Industrial Disease and Dependents Section of the Adjudication Branch, the total number of claims for non-malignant respiratory diseases (including infectious disease) received by the Board in 1981 was 682, similar to the 1980 total of 696. Data indicates that there have been an average of 86 "silicosis" claims received annually by the Board over the last few years. As was the case with cancer, not only does the number of compensated cases of respiratory disease seem to fall far short of the estimate of the prevalence of work-related respiratory illness, but the number even reported to the Board is but a tiny percentage of what it could be. While the goal is clearly to eliminate these hazards from the workplace, all indications are that this situation is still a long way from being reached. Hence the discrepancy between the possible magnitude of work-related factors in respiratory diseases in the Ontario population and the number of occupational respiratory disease cases recognized by the WCB is of potential concern.

There seem to be a number of reasons for the possible discrepancy. Expanding existing identification and reporting systems is not likely to significantly narrow the gap and is somewhat controversial for human rights reasons. Greater outreach efforts may prove useful to some extent. Nonetheless, by far the major issues are clearly related to policy decisions as to what constitutes an occupational disease. As will be described in subsequent sections, difficulty exists often due to the presence of confounding factors, notably cigarette smoking for chronic bronchitis and emphysema, and to a lesser extent pre-existing conditions, notably atopy in a situation of potentially occupationally-induced or aggravated asthma. Administrative factors such as the long delay period may also play a role in discouraging potentially worthy claims and will be addressed below as well.

1.1.2 Facts and Figures Regarding Decisions on the Allowance or Rejection of Respiratory Claims

High Rejection Rate: Although official rejection figures are not kept by the WCB, various studies have been carried out and lists have been kept which suggest that the rejection rate of claims for respiratory disease is very high. As shown in Table IIIB-5 the sample study conducted for this Inquiry revealed that 59% of claims listed in the silicosis index were rejected. It should be noted, however, that this does not mean that 59% of claims for veritable silicosis were rejected, as often the real diagnosis proved to be other than silicosis. In fact, on further breakdown

Table IIIB-5

Allowed vs Rejected Non-Maligant Respiratory Disease Claims at the Ontario WCB According to Various Sources

Source	Claims Included	Total	No. Allowed	%[a]	No. Rejected	%[a]	Other Status
Industrial Disease & Dependents Section	All Chest Disability Reported in 1981[b]	682	311	60	205	40	166[c]
Sample Survey of Silicosis Claims (for this Inquiry)	Claims in Silicosis Index Set-up Just Prior to July 1979	150	57	41	81	59	12[d]
	Claims with Diagnosis of Silicosis or Other Pneumoconiosis	81	43	53.1	38	46.9	-
	Claims with Diagnosis of Other Respiratory Conditions	56	8	14.3	48	85.7	-
Sample Survey of Asbestosis Claims (by Peter Barth)	Claims for Asbestosis Only. Set-up Between 1976-1980	101	47	46.5	54	53.5	-
COLD in Workers Exposed to SO_2 (list kept by Chest Disease Consultant)	All Claims for COLD Adjudicated under SO_2 Guidelines Only	30	17	65	9	35	4[e]

Notes

a. Percent is calculated as Percent of Allowed Plus Rejected. Claims with "other" status were excluded from the total.
b. Excluding cancer, but including infective diseases of the chest and acute respiratory illness.
c. Pending.
d. Claimant did not reply, therefore claims dropped.
e. Claim Status not recorded.

by those diagnosed as silicosis or other pneumoconiosis as compared to those with other respiratory conditions, 38 of 81 claims in the former category (46.9%) were rejected, as compared to 48 of 56 claims (85.7%) in the latter group. Since only a small fraction of chronic obstructive lung disease claims are likely to find their way into the silicosis index, and many fewer of these claims allowed, the rejection rate for respiratory disease claims as a whole (excluding malignancies and infections) is undoubtedly much higher than 59%.

For those workers with chronic obstructive lung disease and documented sulphur dioxide exposure, the only circumstance for which guidelines concerning chronic obstructive lung disease exist, 9 of 26 claims recorded by a Board physician specializing in respiratory disease claims, i.e. 35%, were rejected. Claims for chronic obstructive lung disease not related to sulphur dioxide exposure are seldom allowed. Recall that only five workers with chronic obstructive lung diseases received compensation in 1980. Thus, the existence of guidelines again is shown to be of importance in facilitating the allowance of claims. In Barth's review of 101 asbestosis claims, he found 54 rejections (53%). He noted that 27 of these were rejected by Board physicians who screened them without referral to the Advisory Committee on Occupational Chest Diseases (ACOCD) and 27 were rejected by the ACOCD. (The former group of claims likely lacked evidence to confirm silicosis or other pneumoconiosis as will be explained below.)

Of the 682 claims for non-malignant chest disability (including infective), 311 were allowed and 205 (40%) were rejected. (As the infective disease claims are largely acute and would therefore tend to be more easily recognizable as work-related, inclusion of this group may account for the higher allowance rate compared to other sources.)

Thus, evidence from various different sources confirms that the rejection rate for chronic respiratory disease claims is fairly high, much greater than the 2% rejection of claims overall. This may be a factor discouraging the claimant from even filing a respiratory disease claim at the Board, and in itself indicates the very different nature of the adjudication of these claims as compared to industrial accident claims. (Interestingly, the participation of a union, M.P., M.P.P. or injured workers' group did not seem to significantly affect the outcome of the claim. As shown in Table IIIB-6, 35 of 49 silicosis claims (71%) were rejected as compared to 51 of 101 silicosis claims (50%) in which there was no outside group involvement. This was consistent with the data obtained for cancer claims; for possible interpretation of these data, see Chapter II Part B, Section 2.)

The Delay Factor: The other factor potentially discouraging reporting and indicative of the different nature of these claims is, as was the case for cancer, the long delay period between claim set-up and initial decision. Again no official statistics are kept for respiratory diseases as a whole, but the sample survey from the silicosis index revealed that the mean time from the date the

Table IIIB-6

Count of Claims Received for Silicosis by Outside Group Involvement and First Claim Status[a]

Outside Group Involvement (Union, M.P. or M.P.P. Injured Workers' Consultants, Other)	First Claim Status				
	Allowed Medical Aid Only	Allowed Compensation and Medical Aid	Claim Rejected	Claimant Did Not Reply	Total
Union Only	2	5	24	2	33
Union or M.P. or M.P.P.	-	-	3	-	3
Union & Injured Workers' Consultants	-	-	-	-	-
Union & Other	-	-	-	-	-
M.P. or M.P.P. Only	-	2	3	-	5
M.P. or M.P.P. and Other	-	1	1	-	2
Injured Workers' Consultants	1	-	2	-	3
Other	-	1	2	-	3
Sub-total	3	9	35	2	49
Outside Group Involved	8	31	51	11	101
Total	**11**	**40**	**86**	**13**	**150**

Notes

a. This sample study includes only "silicosis" claims set up prior to July 1, 1979.

Table IIIB-7

Frequency Distribution of Claims Received for Silicosis by Weeks from Date Claim Set Up to Date Initial Decision Reached, and First Claim Status[a]

Calendar Weeks to Initial Decision	Allowed Medical Aid Only	Allowed Compensation and Medical Aid	Rejected Claims	Claimant Did Not Reply	Total Claims	Cum. %
0	-	-	-	-	-	0.0
0 to 1	-	-	-	-	-	0.0
1 to 2	1	-	-	-	1	0.7
2 to 3	-	-	-	-	-	0.7
3 to 4	-	-	1	-	1	1.4
4 to 5	-	-	2	-	2	2.7
5 to 6	-	-	1	-	1	3.4
6 to 7	-	-	2	-	2	4.7
7 to 8	-	-	4	-	4	7.4
8 to 9	-	1	3	-	4	10.1
9 to 10	-	1	7	-	8	15.4
10 to 11	-	1	2	2	5	18.7
11 to 12	-	-	1	-	1	19.4
12 to 13	-	-	5	1	6	23.4
13 to 26	3	14	34	6	57	61.4
26 to 52	5	17	15	3	40	88.1
52 to 104	2	6	8	1	17	99.4
104 to 156	-	-	1	-	1	100.0
Total	**11**	**40**	**86**	**13**	**-**	**150**
Mean Weeks	33.6	31.0	24.5		26.9	
S.E.M.	5.8	2.7	2.0		1.6	
Standard Deviation	19.2	16.9	19.9		19.3	

Note

a. This sample study includes only "silicosis" claims set up prior to July 1, 1979.

claim was set up to the initial decision was 26.9 weeks. For allowed claims the mean number of weeks was even higher, 32.6 weeks, i.e. more than 7-1/2 months. (See Table IIIB-7.) The allowed claims take longer largely because claims may be rejected without even being referred to the ACOCD, thereby avoiding the delay attributable to the additional consultation. (The fact that Board employed physicians can prevent claims from proceeding to the Advisory Committee on Occupational Chest Diseases has been identified as a contentious issue. This and other problems identified in claims procedure are discussed below.)

Table IIIB-8 shows the frequency of appeals and the length of time from referral to appeal until a decision is reached. It was noted that 14 of the 150 claims in the silicosis sample underwent single appeals and two claimants made more than two appeals. For 11 of the claims with single appeals, the mean number of weeks in the appeal process was 31.6 weeks. According to the sample, it took an average of 1.7 weeks to notify the claimant of the initial decision (See Table IIIB-9). Even assuming that the appeal was launched immediately, the fact is that it takes well over a year from the time the claim is set up to the time the claimant receives a decision if he or she chooses to appeal. Thus, the delay factor is not negligible.

The Appeal Process: It should be further noted that, as was the case with cancer claims, appeals largely do not change the outcome. In only one of the 16 silicosis claims that went to appeal was a rejection changed to an allowance (see Table IIIB-10). Given this statistic, the credibility of the appeal system has been questioned. Interestingly, there seems to be a high correlation between outside group involvement and whether or not a claim is appealed. Eleven of the 35 "silicosis" rejections were appealed (31.4%) when a union, M.P., M.P.P. or injured workers' consultant was involved, as compared to 7 of 51 rejections (13.7%) not handled by outside groups. (The cause-effect relationship here can only be speculative.) In the case of the 1 rejected claim that was allowed on appeal, the union and an M.P. or M.P.P. participated. (See Tables IIIB-6 and IIIB-11.)

In summary, there is reason to suspect that occupational factors play a larger role in respiratory disease than is currently reflected in compensation board statistics in Ontario or elsewhere. The rejection rate of respiratory disease claims is significantly higher than the rejection rate for claims overall, which may be a major factor discouraging the reporting of respiratory disease cases in which work-place factors may have been contributory. The delay factor is also problematic not only as it may discourage potentially eligible workers from applying, but because "justice delayed is justice denied". Although appeals contribute markedly to this lengthy procedure, they rarely seem to change the outcome. These facts are indicative of the difficulties encountered in the adjudication of these claims by the present procedure.

As the figures themselves suggest, the procedure for determining the work-relatedness of respiratory diseases is indeed problematic. Moreover, the content of the policy tends to be based on questionable

Table IIIB-8

Frequency Distribution of Initially Rejected Claims by Weeks From Appeal Referral Date(s) to Decision Date(s), Industrial Disease Category and Number of Appeals in Claim

Weeks in Appeal Process	Cancer Appeals in Claim		Silicosis Appeals in Claim		Industrial Noise Deafness Appeals in Claim		
	Single	Multiple	Single	Multiple	Single	Multiple	Total
0	-	-	-	-	-	-	-
0 to 1	-	-	-	-	1	-	1
1 to 2	-	-	-	-	-	-	-
2 to 3	-	-	2	-	-	-	2
3 to 4	-	-	-	-	-	-	-
4 to 5	-	-	-	-	-	-	-
5 to 6	-	-	-	-	-	-	-
6 to 7	-	-	-	-	-	-	-
7 to 8	-	-	-	-	1	-	1
8 to 9	-	-	-	-	-	-	-
9 to 10	1	-	-	-	1	-	2
10 to 11	-	-	-	-	1	-	1
11 to 12	-	-	-	-	-	-	-
12 to 13	-	-	1	-	-	-	1
13 to 26	-	-	2	1	1	-	4
26 to 52	3	-	5	1	-	-	9
52 to 104	4	-	1	-	-	1	6
Appeal Not Completed	5	1	3	-	2	-	11
Total	**13**	**1**	**14**	**2**	**7**	**1**	**38**
No Appeal	47		70		38		155
Mean Weeks^{b}	54.4		31.6	25.8	9.6		35.5
S.E.M.	9.9	-	7.0	9.1	3.1	-	5.3
Standard Deviation	28.1	-	23.3	12.8	7.0	-	27.3

Note

a. This sample study includes only claims set up prior to July 1, 1979. The small numbers in this sub-sample suggest results should be interpreted with caution.

b. Calculated for completed Appeals only.

Table IIIB-9

Frequency Distribution of Claims Received for Silicosis by Weeks from Date Initial Decision Reached to Date Initial Decision Conveyed to Claimant, and First Claim Status

Calendar Weeks to Initial Decision	Allowed Medical Aid Only	Allowed Compensation and Medical Aid	Rejected Claims	Claimant Did Not Reply	Total Claims	Cum. %
0	7	5	16	7	35	23.3
0 to 1	4	19	64	5	92	84.6
1 to 2	-	3	3	-	6	88.6
2 to 3	-	1	-	-	1	89.3
3 to 4	-	3	-	-	3	91.3
4 to 5	-	5	2	-	5	94.6
5 to 6	-	2	1	-	3	96.6
6 to 7	-	-	-	-	-	96.6
7 to 8	-	1	-	-	1	97.3
8 to 9	-	-	-	-	-	97.3
10 to 11	-	-	-	-	-	97.3
11 to 12	-	-	-	-	-	97.3
12 to 13	-	-	1	-	1	98.0
13 to 26	-	1	-	-	1	98.7
26 to 52	-	-	-	-	-	98.7
53 to 104	-	-	1	1	2	100.0
104 to 156	-	-	-	-	-	100.0
Known	-	-	-	-	-	100.0
Total	**11**	**40**	**86**	**13**	**150**	
Mean Weeks	0.1	2.1	1.7		1.7	
S.E.M.	0.0	0.5	0.8		0.6	
Standard Deviation	0.2	3.2	8.3		6.9	

Note

a. This sample study includes only "silicosis" claims set up prior to July 1, 1979.

Table IIIB-10

Count of Initially Rejected Claims Received for Silicosis By Appeal Process and Last Outcome[a]

	Last Outcome in Appeal Process				
al Process	Rejected Claim Allowed	Other	No Change	No Appeal	Total
dicator - No Hearing	-	1	1	-	2
dicator - With Hearing	-	1	4	-	5
al Board Only	1	2	4	-	7
dication - With Hearing Appeal Board	-	-	2	-	2
ppeal	-	-	-	83	83 (70)[b]
l	**1**	**4**	**11**	**83**	**99 (86)[b]**

s

This sample study includes only "silicosis" claims set up prior to July 1, 1979.

Actually, there were only a total of 86 rejections plus 13 claims in which the claimant did not reply. Of the 86 rejections, 70 did not go to appeal.

Table IIIB-11

Count of Claims Received for Silicosis by Outside Group Involvement and Last Appeal Outcome[a]

Outside Group Involvement (Union, M.P. or M.P.P., Injured Workers' Consultants, Other)	Last Outcome From Appeal Process						
	No Appeal Made	Appealed	Rejected Claim Allowed	Benefits Increased	Other	No Change	Total[b]
Union Only	27	-	-	-	1	5	6/33
Union & M.P. or M.P.P.	-	-	1	-	2	-	3/3
Union & Injured Worker Consultants	-	-	-	-	-	-	-
Union & Other	-	-	-	-	-	-	-
M.P. or M.P.P. Only	4	-	-	-	-	1	1/5
M.P. or M.P.P. and Other	2	-	-	-	-		2
Injured Workers' Consultants	3	-	-	-	-	-	3
Other	2	-	-	-	-	1	1/3
Sub-total	38	11	1	-	3	7	10/49
No Outside Group Involvement	94	-	-	-	1	6	7/101
Total	**132**	**11**	**1**	-	**4**	**13**	**150**

Notes

a. This sample study includes only "silicosis" claims set up prior to July 1, 1979.

b. The first number (where 2 are listed) represents the total number that were appealed. The second number represents the total number handled by the group specified.

assumptions, as will be decribed in detail in subsequent sections. Issues of concern were found to be similar to those identified in Chapter II (with respect to cancer) and include the lack of widespread input in the development of policy or even awareness of the topics being considered, and the heavy burden of proof requirement on which the policy is based. As was shown to be the case with cancer claims, the negative hypothesis is assumed until the affirmative is proven.

It must also be borne in mind that a major reason for difficulties encountered relates to the presence of confounding factors: smoking, pre-dispositions or pre-existing conditions. These effect the identification and reporting of workers potentially eligible for compensation, the various policy decisions that have been established and the adjudication of specific claims.

1.2 Disability Assessments

In the sample survey, the permanent disability (P.D.) award for claims in the silicosis index ranged from 1% to 100% with an average P.D. award of 23%. As shown in Table IIIB-12, the largest number of silicotics received a 10% permanent disability award. (The Board usually uses multiples of 10 in the assessments.) One allowed claim for chronic obstructive lung disease crept into this index and received a 30% permanent disability award, one asthma claim received a 20% permanent disability award. As will be illustrated in Section 2.3 below, these assessments have been the source of considerable discontent. It is therefore worthwhile examining the issues surrounding the assessment of disability and impairment in workers' compensation.

1.2.1 Basis of Disability Awards:

The determination of the size of a disability award is complex and based on a multitude of assumptions, only a few of which are in the domain of the medical profession. As discussed in Section 4.1 of Part A of this chapter, first it must be determined on what basis the compensation is being given. Is it to compensate for a physical impairment that resulted from workplace conditions or is it to compensate for the economic impact of that impairment?

Secondly, even if the impairment model is chosen, it must be decided how "impairment" is to be defined: Is it to be based on the extent of deterioration from one's previous capabilities (over and above the aging effect) or is it to be defined as the extent of deviation from "normal"? As also discussed in Part A above, how "normal" is defined is, in fact, also problematic.

Thirdly, it must be decided which parameters are to be used to measure impairment and how they are to be weighted: should respiratory impairment be based on pulmonary function tests alone? To what extent should chest x-ray findings be taken into account? Should subjective symptoms be considered important in the assessment? As pointed out in Section 4.1 above, different institutions have chosen different combinations of factors.

Table IIIB-12

Frequency Distribution of P.D. Claims in the Silicosis Index by Diagnosis and % P.D. Award to Date[a]

	Percent Permanent Disability Award to Date												
Current Diagnosis	1.0 4.9	5.0 9.9	10.0 19.9	20.0 29.9	30.0 39.9	40.0 49.9	50.0 59.9	60.0 69.9	70.0 79.9	80.0 89.9	90.0 99.9	100%	Total
Silicosis	1	-	19	8	5	-	3	-	-	-	-	1	37
Tuberculosis	-	-	-	-	-	-	1	-	-	-	-	-	1
Pneumoconiosis	-	-	1	-	-	1	-	-	-	-	-	-	2
Chronic Obstructive Lung Disease	-	-	-	-	1	-	-	-	-	-	-	-	1
Bronchial Asthma	-	-	-	1	-	-	-	-	-	-	-	-	1
Total	**1**	**0**	**20**	**9**	**6**	**1**	**4**	**-**	**-**	**-**	**-**	**1**	**42[b]**

Note

a. Sample Survey of 150 Claims Set Up prior to July 1, 1979

b. Two other allowed claims were found in the silicosis sample, 1 for mesothelioma and 1 for noise-induced hearing loss accounting for the total of 44 allowed P.D. claims referred to in other tables.

The fact that no one system has been applied universally is indicative of the controversy in this area. Section 4.2 introduced the uncertainty with respect to the significance of chest x-ray findings, and illustrated the considerable inter-reader variability in chest x-ray readings. Section 4.3 above illustrated the difference in opinion as to which pulmonary function tests are the most sensitive indicators as well as the problems in interpretation of these tests. Section 4.4 showed that objective measurements do not necessarily correlate with subjective symptoms and discussed the various factors that influence symptomatology. The extent to which these factors should be taken into account, were, in turn, shown to be matters of policy rather than medical science.

In Ontario, the disability decisions are made by physicians (the Advisory Committee on Occupational Chest Diseases for the pneumoconioses, and Board-employed physicians who specialize in chest disease claims for the obstructive lung diseases). These assessments are primarily based upon an impairment model, but allow for "other factors" to come into play. No written guidelines exist for disability decisions and the basis on which they are derived in individual cases is not spelled out in the claim file. (In fact, pulmonary function test results are not even always listed.) Moreover, the reasons for these decisions are not conveyed to the claimant and lead to much misinterpretation and distrust, as will be illustrated below.

1.2.2 Disability Awards in the Presence of Non-Compensable Disability:

A fourth policy decision that must be made is whether or not the physical impairment resulting from a compensable disease should be isolated from pre-existing or concomitant impairments, either of the same organ system or elsewhere in the body. The medical aspect to this question relates to the accuracy with which this can be done. The difficulty associated with separating the effects of chronic obstructive lung disease from those of silicosis was reviewed in Section 2.4 of the preceding part of this chapter. It was pointed out that although chronic obstructive lung disease produces an "obstructive ventilatory defect", whereas silicosis tends to be thought of as producing "a restrictive ventilatory pattern", to the extent that any significant pulmonary function abnormalities occur, obstructive changes do indeed develop in silicosis. Pulmonary function impairment in an individual with chest x-ray findings suggestive of silicosis, in fact, is often attributable to associated chronic obstructive lung disease. The extent to which the impairment due to non-compensable chronic obstructive lung disease is aggravated by impairment attributable to silica exposure is admittedly difficult to measure, but nevertheless does not seem to be taken into account in determining the disability award.

Since no written policy exists on this subject, and little explanation of the disability assessment is provided in the claim files, it is difficult to be certain exactly what the practice is. It does, however, seem to be inconsistent with the policy and practice of the Board with respect to injury due to accident.

According to the Guidelines for Claims Adjudication involving pre-existing conditions..."The presence of a pre-existing condition is reflected in any permanent disability award when the degree of residual disability found in such cases is increased owing to the underlying condition...." Where a prior condition is not measurable but enhances the residual disability resulting from the accident, a formula is applied. If the pre-existing condition produced minor pre-accident disability - full assessment is awarded; if the condition produced moderate pre-accident disability - 75% of total assessment is awarded; if the condition produced major pre-accident disability - 50% of the total assessment is awarded. If this was applied in respiratory cases, even an individual with severe asthma or bronchitis should be entitled to 50% of the total assessed disability. As will be illustrated in Section 2.3 below, this does not happen. Although an accident is a discrete event making it considerably different from a disease which develops insidiously, the lack of symmetry in the approach to "pre-existing" conditions is nonetheless noteworthy.

Ontario's approach to compensating respiratory disease claims also differs from the practice in other jurisdictions. In answer to a questionnaire sent out to all the Canadian compensation boards, Manitoba Workers' Compensation Board, for example, specified that the relationship between compensable and non-compensable impairment would "be deemed to exist where the injury is to the same part of the body or body member where the pre-existing condition is located, and a medical opinion is unable, on a balance of probabilities, to negate aggravation, acceleration, or enhancement of the condition by the injury, whether it be temporary or permanent in nature."

They illustrated this policy as follows:

> "We have had, for example, the case of an individual suffering from silicosis and emphysema. It was determined that the degree of disability for permanent partial disability attributable solely to silicosis was 10%, whereas a man's overall disability, including his emphysema, was 30%. In this case, the pre-existing condition plays a major role in determination of his present disability, and we would allow a minimum award of 50% of the pre-existing disability, i.e. 50% of the 20% attributable to emphysema, and, of course, to that the 10% compensable award for silicosis, which would give the worker a total entitlement of 20%."

The Manitoba Board also provides for the augmentation of a pension award above and beyond clinical rating, as an individual measurement of loss of earning capacity.

> "In another example, a 52-year-old miner filed a claim for silicosis, due to his employment as an underground miner. Several years before he had a left pneumonectomy for a squamous cell carcinoma. A silicosis panel rated his disability due to silicosis at 25%,

> but determined that this, added to his pre-existing condition, rendered him totally disabled and unable to work. The worker was given an award based upon 25% for silicosis disability and one-half of 75%, representing payment of the portion of the pre-existing condition which our policy prescribed. In other words, a total of 62.5%."

Other jurisdictions use different approaches. In short, there is no unanimity as to the best way to approach this difficult issue, which clearly is a question of policy rather than scientific judgement.

1.2.3 Disability Awards for Multifactorial Diseases:

A fifth policy decision that must be made involves diseases of multifactorial etiology. The difficulty associated with trying to determine how much of a case of chronic bronchitis could be attributed to the workplace as opposed to smoking was discussed at length in Section 3.2 above. It was noted that studies suggest that certain characteristics of industrial bronchitis, such as the predominantly large airway effect, tend to distinguish it from bronchitis associated with cigarette smoking, in which the smaller airways are often affected first and emphysematous changes are more common. However, there is considerable controvesy in attempting to apply these gross generalizations to individual cases, as will be illustrated in Section 3.3.2 below.

Even when the Board does acknowledge the role of workplace factors in chronic obstructive lung disease, the formulae chosen by the Board to apportion causation are indeed questionable. As will be discussed in Section 3.3.1 below, under the sulphur dioxide guidelines, only 20% of the assessed disability in smokers and 40% in non-smokers is deemed work-related. There is little logic to this formulation. For example, if only 40% of the disability from chronic bronchitis in a non-smoker is attributed to the workplace, what accounts for the other 60%?

No other jurisdiction that responded to the questionnaire attempted to apply apportionment of causation in this manner. As stated in Alberta's policy:

> "When a worker suffers a non-malignant chest disease due in part to occupational and in part to non-occupational factors, the overall disability will be presumed to be related to the employment and compensation awarded accordingly...The portion of the award related to non-occupational factors is charged to the Reserve for Enhanced Disabilities."

It should be noted that Ontario does not attempt to apportion causation in cancer claims. Although both occupational and non-occupational factors have an effect on whether or not a tumour will develop (see Chapter II Part A), the policy is to either allow or reject each claim in an all-or-none fashion (see Chapter II Part B).

Table IIIB-13

Silicosis Claims, Set Up Prior to July 1, 1979 by Compensation Benefit Paid or Allowed

Compensation Benefit Paid or Allowed	Silicosis
Temporary Total Only (TT)	2 [a]
Permanent Disability (P.D.)	35
Death Claim Allowed	-
T.T. and P.D. Benefits	9
T.T. and Fatal Benefits	2
P.D. and Fatal Benefits	- [a]
T.T., P.D., and Fatal Benefits	-
No Compensation Benefits for disability or death	102
Total Claims	**150**

Note

a. By the date of completion of this survey, November 1981

1.2.4 *Disability Assessments in the Presence of Genetic Pre-Dispositions:*

The apportionment of causation outlined in the sulphur dioxide guidelines is not applied in other cases of non-malignant respiratory diseases in Ontario. However, the formula for compensating emphysema claims seems even more peculiar. The Board has decided that only those workers with a specifically identified genetic predisposition to develop emphysema will be compensated if the disease occurs on exposure to a variety of occupational settings that have been linked with the disease. As will be explained in Section 3.2.1 below, the guidelines for alpha-1-antitrypsin deficiency emphysema state that 50 - 100% of the assessed disability should be awarded if the claimant is a non-smoker, and 25 - 50% if the claimant smokes, according to his or her genetic characteristics.

In contrast, for asthma claims, the presence of a genetic pre-disposition (atopy) is a disadvantage with respect to compensation, and results in the allowance of benefits for only the acute episodes directly attributable to the workplace. This is similar to accident claims wherein compensation ceases when the employee's condition has reached the pre-accident stage. In accident claims, however, if disability is prolonged due to the pre-existing condition the claimant is still entitled to benefits (although the cost may be transferred to the Second Injury Enhancement Fund). In contrast, if an atopic worker is no longer able to continue at his or her job, i.e. is disabled as a result of the asthma that occurs at work, the fact that a pre-disposition existed will result in the cessation of benefits, even if the individual was asymptomatic prior to the work exposure. This will be illustrated in Section 3.1 below, and constitutes yet a sixth policy decision, that is, whether or not an "inherited predisposition" should be allowed to negate entitlement to benefits or rehabilitation.

In this regard, the Honourable Mr. Justice Tysoe in his Report on Workers' Compensation in British Columbia noted "that 'pre-existing disability' and 'pre-existing condition' are not synonymous. 'Condition' is a general word, and, if there are other things with it, it may constitute a disability. It is 'disability' that counts in Workers' Compensation and not mere 'condition'." An amendment was therefore made in response to Tysoe's recommendation, specifying that "the mere fact that the worker suffered from some weakness, disease, or vulnerability which partially caused his personal injury or disease is not sufficient to bring proportionate entitlement into operation. The pre-existing condition must have amounted to a disability prior to the occurrence of the injury or disease." Although "proportionate" entitlement is not actually invoked in Ontario where "pre-existing condition" is found, the latter does indeed affect the eligibility to workers' compensation benefits.

The implied inconsistency in the role of genetic predisposition with respect to emphysema as compared to asthma claims may relate in part to the fact that asthma is characterized by episodic exacerbations with the possibility of no real disability between bouts, however, emphysema is characterized by irreversible damage, hence it would be impossible to administer a policy of compensating only the specific exacerbations in question. Nonetheless, the denial of compensation to those without the appropriate genetics, as illustrated in Section 3.2.2 below, is certainly anomolous. Moreover, the fact that the question of genetic predisposition comes into play at all is contrary to the spirit of "taking a worker as he or she is found".

1.2.5 Entitlement to Wage-Loss Benefits:

Controversy over fitness to return to work after an injury has always existed in workers' compensation systems. In the area of chronic respiratory diseases relatively few claims involve total temporary disability (with the exception of asthma, the acute component of which does not really apply to this discussion). As shown in Table IIIB-13, in the 150 claims from the silicosis index reviewed in the sample study, 48 (32%) of the claimants received compensation awards, but only 13 (27%) of these (less than 9% of the total) ever qualified for total temporary benefits. These included two claimants who received total temporary benefits only, 9 claimants who received total temporary benefits prior to a permanent disability award and two claimants who received total temporary benefits prior to fatal benefits. Thus 35 of the 48, i.e. 73% of the successful claimants, never qualified for off-work benefits. Only 1 of the permanent disability awards was a 100% disability assessment, which, in fact, was the only one of the 35 whose permanent disability award was greater than 50%. (See Table IIIB-12).

It is not known how many of these 35 claimants were unable to work and how many remained on the job, but clearly a seventh policy decision is how the causation problem with respect to wage-loss benefits is to be decided. The 1981 White Paper concerning workers' compensation reform recommended a lump sum award for physical impairment in addition to entitlement to wage loss benefits which would be paid to the extent that the workers compensable condition was judged responsible for his or her economic disability. Obviously the question of what constitutes work-relatedness of disability takes on immense importance.

An eighth policy decision that must be made relates to the eligibility criteria for total temporary or wage loss benefits both in the period of diagnostic work-up (for example, occupational asthma) and in the period after the diagnosis has been confirmed and relocation away from exposure is necessary or desirable. As described in Part A, for certain situations, such as the development of sensitivity to a substance, the necessity of relocation is recognized. For silicosis or "pre-silicosis", it was stated that the evidence that removal from exposure will affect the progression of the disease is equivocal, although it is clearly a necessity in advanced cases to prevent complications. For chronic obstructive lung diseases, there is certainly a strong clinical impression that removing the worker from dust exposure at least improves his or her symptoms, but the ultimate effect on prognosis has not been documented. For asbestos, the issue is complicated by the fact that asbestos is a known carcinogen and replacing one worker with another serves to increase the population at risk which is certainly ill-advised.

In summary, data have been presented and issues identified both in relation to the work-relatedness of a certain impairment and to the assessment of disability attributable to this condition. It was stated that occupational respiratory diseases are probably being under-compensated in Ontario or more specifically, occupational factors may be playing a role in a great many cases of respiratory disease, presently not recognized as such. This relates mainly to policy decisions regarding what constitutes an "occupational" disease, specifically due to the many confounding factors, notably smoking. The role of genetic factors has also been identified as problematic. Disability assessment was discussed in Part A of this chapter and the controversy about facilitating the removal of a worker with respiratory impairment from additional hazardous exposure was also canvassed. The difficult problem of determining the extent to which the occupational component of a worker's disability contributes to his or her inability to work was also introduced.

The next two sections will describe the evolution of the Board's policy on the adjudication of respiratory disease claims and will provide case examples to illustrate the issues identified.

2. The Adjudication of Pneumoconiosis Claims

2.1 Evolution of Policy

The pneumoconioses were first recognized as industrial diseases in Ontario in 1926 and the policy of the Workers' Compensation Board on the adjudication of these claims has evolved significantly since that time. The only disease which is specifically scheduled in the Act, with the consequent rebuttable presumption in favour of the worker, is silicosis. However, the inclusion of "pneumoconioses" per se later that year gave the Board the authority to allow claims for asbestosis or any other pneumoconioses it deemed appropriate.

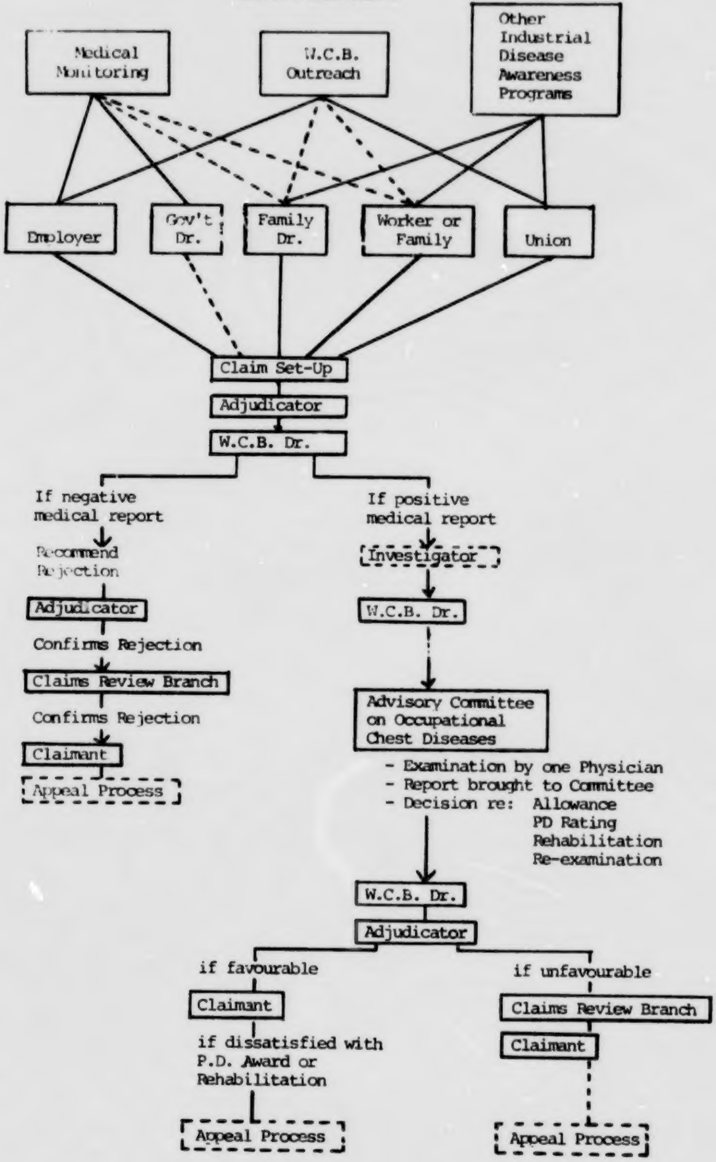
In 1942 the first claim for asbestosis was awarded. Claims have also been allowed for bauxite pneumoconiosis (75 cases), berylliosis (3 claims), graphite pneumoconiosis (2 or 3 claims), nepheline syenite pneumoconiosis (8 or 9 claims) and a few claims for talcosis, cadmium pneumoconiosis, hard metal disease, silo-fillers disease, and various organic (vegetable or animal) dust diseases including bagassosis, bird handler's lung, farmer's lung, etc.

Stipulations in the Act as to the required period of exposure evolved and presently a two-year history of exposure for silica dust, for example, is required. The initially stipulated requirement that the claim be filed within a year of exposure was altered and finally dropped completely in 1944. (It should be noted that in some jurisdictions a time limitation on filing a claim still prevails.) The definition of the disease "silicosis" was also altered as time progressed.

Fig. IIIB-1

Claims Adjudication - Industrial Diseases

SEQUENCE OF EVENTS


Medical Monitoring
W.C.B. Outreach
Other Industrial Disease Awareness Programs
Employer
Gov't Dr.
Family Dr.
Worker or Family
Union
Claim Set-Up
Adjudicator
W.C.B. Dr.
If negative medical report
Recommend Rejection
Adjudicator
Confirms Rejection
Claims Review Branch
Confirms Rejection
Claimant
Appeal Process
If positive medical report
Investigator
W.C.B. Dr.
Advisory Committee on Occupational Chest Diseases
- Examination by one Physician
- Report brought to Committee
- Decision re: Allowance
PD Rating
Rehabilitation
Re-examination
W.C.B. Dr.
Adjudicator
if favourable
Claimant
if dissatisfied with P.D. Award or Rehabilitation
Appeal Process
if unfavourable
Claims Review Branch
Claimant
Appeal Process

Problems Identified

1. Recognition and Reporting of Cause
2. Role of Treating Physician
3. Response of W.C.B. Dr. to Medical Report
4. Assessment of Exposure
5. Role of Adjudicator
6. Role of Claims Review Branch
7. Role of Advisory Committee
8. Letters to Claimant
9. P.D. Award
10. Rehabilitation
11. Appeal Process

Peter Barth, in reviewing the adjudication procedure for asbestosis claims in a study for the Royal Commission on Asbestos, noted that the only procedural guidelines that applied were, first the need for a "clear and adequate history of occupational exposure to asbestos" and, secondly that there must be a "diagnosis of frank asbestosis." He commented on the lack of precision of the "clear and adequate history" and noted that the burden of judgment simply lodged with the Advisory Committee to render a decision on the case. Detailed guidelines do, however, exist on asbestos fiber dust effect as eligibility for rehabilitation for asbestos-exposed workers is largely determined by these specifications.

The Advisory Committee on Occupational Chest Diseases originated in 1927 shortly after the pneumoconioses were recognized. Its initial name "The Silicosis Referee Board" was changed in 1975 to better reflect its role. Although its mandate was clarified on several occasions, neither its actual membership nor its role has altered significantly. Some problems relating to the functioning of the Advisory Committee on Occupational Chest Diseases will be discussed in the next section.

Medical monitoring facilities were established in Ontario in 1928 and a number of administrative changes have taken place over the years. The role of medical monitoring in the identification of victims of pneumoconioses was alluded to earlier.

Rehabilitation for workers affected by pneumoconioses first began in 1975 with a rehabilitation program for Elliot Lake miners. The expansion of this program in the following year to include asbestos workers and others affected by industrial diseases has met with limited success, as will be mentioned in the next section.

The chronology of key events in the development of the Workers' Compensation Board policy on the adjudication of silicosis and other pneumoconiosis claims is outlined in Appendix 1.

2.2 Claim Procedure in the Adjudication of Pneumoconioses

Once a claim is set up it is controlled by a Claims Adjudicator in the Industrial Disease and Dependents Section. A decision is made as to which employers need to be sent a Form 7S. There may be significant delay as exposure may have occurred at more than one company and as these are generally long latency diseases, some of the companies may even have gone out of business. Once the Form 6S from the worker, 7S from the company and 8S from the doctor have been accumulated, the Adjudicator sends the information to the Medical Division for an opinion.

Based on this information, Board-employed physicians have the authority to either reject the claim or to send an investigator to assess workplace exposure. Further medical documentation may be sought and even the opinion of a pathologist may be solicited. According to Barth, this may result in a delay of six weeks to six months. Alternatively, the Board doctors may decide that the claim may well be successful and hence merits the attention of the Advisory Committee on Occupational Chest Diseases. This Committee then decides if the claim is to be allowed, and, if so, at what percentage of disability. Eligibility for rehabilitation and date of re-examination are also determined. Once these decisions are reached by the Advisory Committee, they are conveyed back to the

Board-employed physicians who referred the claim to the A.C.O.C.D. and, in turn, to the adjudicator.

If the decision is adverse to the claimant, it is referred to the Claims Review Branch, which we have found invariably confirms the rejection in these cases and then conveys the decision to the claimant. If the decision is favourable, the adjudicator informs the claimant directly. The decision is open to appeal; first to an appeal adjudicator and finally, to a three-person panel of Appeal Commissioners. The Ombudsman may be used if dissatisfaction with the outcome of an appeal persists. If new information becomes available, a request for re-consideration may be made. In this sense, the case is never closed.

The sequence of events in the life of a claim is shown diagramatically at page 415. On the right is a list of some of the major problem areas identified in the process. These eleven points will be elaborated upon briefly below.

1. The identification of workers with occupational respiratory diseases: The outreach program of the Board with respect to asbestosis was alluded to above. The Board does periodically hold seminars, send letters or write articles addressed to the medical profession regarding industrial diseases.

   A number of submissions presented to the Weiler Inquiry, particularly from labour organizations, have referred to the need for more extensive programs to track down victims of industrial diseases. Although much of this concern was expressed specifically in relation to occupationally-related cancer, the Board should be encouraged to continue and expand its outreach program in all areas of industrial diseases.

   The government program of medical monitoring for respiratory diseases was mentioned in Section 1.1.1. With the advent of the Occupational Health and Safety Act, the onus of medical monitoring is transferred directly to the employer. Representatives from the Ontario Mining Association and others have expressed concern that the effective record-keeping that resulted from the Ministry-run chest examination stations may be undermined. Others oppose medical monitoring because it may interfere with the privacy rights of the individual concerning his or her health. A discussion of the uses and misuses of medical monitoring is, however, beyond the scope of this report.

2. The role of the treating physician: A number of workers with respiratory diseases who appeared before the Weiler Inquiry stated that their own doctors were never informed of their diseases, despite x-ray evidence of effects resulting from workplace exposures. On the other hand, it seems that frequently the worker's own doctor tells the worker that he or she has developed an occupational disease, only to find that the Board rejects the claim. In a presentation to this Inquiry, one worker read his x-ray report indicating evidence of asbestos effect and contrasted this with the Board's decision that he did not have any evidence of an industrial disease. Other workers, labelled as "pre-silicotic" by the Board for example, have accused the Board's

doctors of lying to them as other doctors had told them that they had silicosis.

Part of the problem is certainly related to the fact that "pre-silicosis" or "asbestos fiber effect" are not widely recognized clinical entities but rather concepts relied upon by the Board in an attempt to detect the disease prior to its frank manifestation. In all the textbooks and articles reviewed for this report, not one mention of these entities occurred. Some radiological texts, however, do allude to this terminology. (Both terms refer to the situation in which the chest x-ray shows evidence of the occupational exposure, but at the stage before the disease itself has become manifest.) Aside from whether or not the Board is justified in utilizing these concepts, medical practitioners can hardly be faulted for "misleading their patients" when x-ray evidence of dust effect is recognized. (The widespread use of the ILO U/C coding system would alleviate this problem to some extent as it is recognized to be purely a descriptive tool and has no direct implications as to disability.)

The resentment and frustration that arise from conflicting medical opinion is illustrated by a letter that appeared in an article in the British Medical Journal of February 1966 and was cited by a senior Board physician in a seminar presentation. "The Board doctors say that I have bronchitis and cardiac hypertension. Dr. X in hospital says silicosis, Dr. Y, tuberculosis, Dr. Z thinks I have silicosis, you say bronchitis. I've worked as a pottery caster for 40 years and after 14 years of ill health, the Ministry tells me that doctors do not make mistakes." Unfortunately, the many cases reviewed for this Inquiry revealed that this situation occurred frequently.

Board medical staff do, in fact, feel that family doctors often create the biggest problems for the Board by leading their patients to false expectations for compensation and by advising their patients with respiratory diseases to leave a dusty workplace. As Board policy on relocation and rehabilitation would make the vast majority of these workers ineligible for benefits, an adversarial relationship may then develop between the worker and his or her doctor on one hand and the Board on the other. In an address to the medical community in December 1979, one senior Board physician wrote:

> "The Board is concerned that far too frequently patients are advised by their family or consultant physicians to quit the workplace because of perceived disease which, in fact, may not be present, or if so, may be of a degree which would ordinarily not require removal from ongoing exposure. Once advised, such an individual may then present himself to the Board requesting retraining, compensation or pension to find that the Board is unable to act, either because the claim does not meet the requirements or because there is clearly no need for an individual to abandon his trade... It is clear that a physician should never under any circumstances advise his patient to leave his life's work without first

> requesting an opinion from the Workmen's Compensation Board. The Board is only too anxious to investigate and evaluate such a request from any physician on behalf of his patient. The Board's view is that it is difficult for an average physician to accumulate knowledge and experience in respect to mineral dust pneumoconiosis and while advice may be well-meaning, unreasonable expectations in the patient, once aroused, may never be retrieved."

Although better communications between the Board and the medical community as to Board policy as well as industrial diseases generally is certainly desirable, it seems that often the problem is not in communications, but lies in the very nature of the policy of the Board itself. Whereas the primary concern of the family physician should be his or her patient, the Board has other concerns. Recognizing that there is considerable uncertainty whether removal from exposure alters the patient's prognosis, physicians can hardly be criticized for advising their patients to get out of hazardous working conditions regardless of the extent of already established respiratory disease, whether of occupational origin or not. (This issue will be addressed further under point 10.)

3. Asymmetrical pattern of response of the Board doctors: An asymmetrical pattern of response by senior Board physicians to negative as opposed to positive medical evidence of industrial disease has been identified both by Peter Barth for the Dupre Commission, and in reviewing claims for this Inquiry. When a worker's doctor reports that he or she is suffering from occupational pneumoconiosis the report is not considered definitive and is submitted for further consideration. If, however, the worker's doctor reports that he or she is in good health or has an illness attributed to chronic obstructive lung disease likely due to smoking, for example, this is almost routinely accepted at face value and the claim is rejected. The cases presented below indeed illustrate that the Board doctors often cite the fact that the attending physicians did not acknowledge a work-relationship as a reason for rejecting a claim, yet rarely, if ever, do they cite the opinion of the attending physician as a reason for allowing a claim.

   Point 2 above helps explain why this takes place. The Board feels that the worker's doctor is neither objective nor adequately knowledgeable to diagnose pneumoconiosis. Interestingly, although Board consultants contend that doctors are often biased in favour of the worker, there are many publications both in the medical literature and in the lay press which have addressed either the general under-recognition and under-reporting of occupational disease by physicians or have implied that physicians, at least in some communities, were biased in favour of the company. In Tataryn's book, Dying for a Living, for example, he quotes Paul Falkowski, former health and safety co-ordinator of the Steelworkers as follows: "I presume that over the entire period when the effects slowly began showing on the people most doctors in Elliot Lake must have known about this. I have no other choice but to believe many doctors chose to err on the side of the company rather than on the side of the men who were affected." Tataryn also alluded to the comments of Dr. David Bates, Chairman of the Science Council of Canada's investigation of policies and poisons, underlining the less-than-distinguished role of the medical profession on occupational health matters.

In any case, the difficulty in diagnosing these conditions and the generally poor knowledge about occupational diseases in the medical community at large (See Chapter I, Part A - Section 3) certainly could just as easily lead to under-rating the occupational element or missing the diagnosis of an occupational disease as it could to the over-diagnosis of pneumoconiosis. This, therefore, is a powerful argument that negative as well as positive reports from physicians merit careful attention.

4. Assessment of exposure: For the occupationally-specific diseases, the assessment of the workplace takes on less importance than for the non-specific diseases. In the former situation, once it is established that the worker has silicosis or asbestosis, for example, there is no question that it stems from exposure to silica or asbestos. Since silicosis is a scheduled disease under the Act, it is presumed to have arisen from employment in certain specified employments and is therefore compensable. For asbestosis, Barth found generally that few, if any cases are rejected due to failure to prove exposure. The assessment of the workplace in these situations may have relevance with respect to cost-charging and the necessity for relocation of workers, not to mention prevention, however, is not essential in the adjudication of the claim.

On the other hand, for non-specific respiratory diseases such as the chronic obstructive lung diseases, assessment of the workplace takes on great importance. It would be inconceivable to decide on the role of workplace conditions in the development of these diseases without assessing the workplace. Admittedly this is difficult as the exposure may have taken place years earlier, there may have been a number of workplaces involved, the workplaces may have changed considerably, or the company may not be still in business. Frequently, investigators do go out to the workplace, however, this is at the discretion of the Board's medical consultants. On reviewing claims it was found that there was a much greater emphasis placed on accumulating medical information about the individual worker rather than on the accumulating industrial hygiene information on the content and extent of exposures in the workplace.

Although the industrial hygienists from the Ministry of Labour are often called upon in this task, concern has been expressed that far too often a Board investigator with little industrial hygiene training merely questions the employer and a few workers without adequately appraising the workplace. Also, as will be illustrated in some cases presented below, awaiting the Ministry of Labour investigation may create significant delay. Moreover, claims may be rejected without the workplace being assessed at all simply because the occupation in question has not been proven to have a higher incidence of the disease in question. Particularly hazardous conditions in a specific plant may therefore go unnoticed if an increased incidence of disease has not been observed.

5. Role of the adjudicator: Although the adjudicator officially controls the case, it has been repeatedly observed by this author and others who have reviewed the files that the adjudicator almost always accepts the recommendation of the Board's medical consultants. In 156 cases reviewed by Barth, he found only one in which the recommendation of the doctors was not acted upon by the adjudicator. In my own review, only one case was encountered in which the recommendation of the Board's doctor was not followed. In this case the Board doctor recommended allowance, but the adjudicator in consultation with senior Board personnel felt that this would be inconsistent with Board policy and denied the claim. (This case is included in the case illustrations - see Case No. 3, Section 3.2.2.)

6. The Claims Review Branch: Of the 86 silicosis claims that were rejected in the sample survey of 150 silicosis claims, 73 claims went to the Claims Review Branch and 13 did not. Of the 64 allowed claims, 5 were sent to the Claims Review Branch (likely due to dissatisfaction with the permanent disability award). In none of the 78 claims that were referred to the Claims Review Branch was a change made in the original decision. Moreover, in the entire sample survey of industrial diseases (including cancer and noise-induced hearing loss) the Claims Review Branch never changed a single one of 152 rejections that were sent to it. This may be due to the confidence which the Claims Review Branch has in the adjudicators at the Industrial Disease and Dependents Section. More probably, however, it may reflect their reluctance to overturn decisions that were based upon the opinions of Board medical staff. This fact, however, does call into the question the usefulness of this step in the sequence of events, and at present it seems to represent an unnecessary delay.

7. The Advisory Committee on Occupational Chest Diseases: As described in the evolution of Board policy on pneumoconiosis, the Advisory Committee on Occupational Chest Diseases has always been responsible for the most important decisions on pneumoconiosis claims, i.e. whether or not the claim is compensable and if so, at what level. In 1929 when its forerunner, The Silicosis Review Board, was created, it was composed of physicians from the Division of Industrial Hygiene of the (then) Ministry of Health with consultants appointed by the Board from the staff of the University of Toronto. All the original appointees served until 1961 and even since then there has been little turnover. At present, it consists of physicians from the Ministry of Labour or Health, as well as a consultant from the University of Toronto, and the Director of the Occupational Health Program at McMaster University. (Also, a pathologist from the University of Toronto works closely with the Committee.)

Typically one of these physicians examines the claimant (usually at a location other than the Board itself) and reports his findings to the Committee which then evaluates the claim. Minutes of the meetings are not kept, votes are not recorded, and there is no indication if a difference of views existed. Moreover, there is no explanation given as to how the decisions were arrived at or what weight was given to various factors.

Factors that are taken into account include the history, the physical examination, chest x-ray, pulmonary function tests (including FEV_1, FVC, FEV_1/FVC, diffusing capacity and often maximum oxygen uptake with exercise and partial pressure of arterial blood gases). It seems that the chest x-ray is indeed heavily relied upon to determine the presence or absence of disease, and the pulmonary function tests play a major role in defining the level of impairment. However, no guidelines are specified, and no impairment rating schemes exist on which the percentage impairment ratings are based. It may be assumed that the recommendations of the Task Force on Occupational Respiratory Diseases (Pneumoconiosis) serve as a rough guideline, although certainly this is not invariably true. (An obvious inconsistency with the recommendations of the Task Force is the persistent failure of the WCB or the Advisory Committee to use the ILO U/C classification of chest x-rays.)

Although apparently after the acquisition of their newest member some differences in the pattern of decisions did begin to emerge (for example, workers with clear x-ray evidence of silicosis began to receive compensation even in the presence of "normal" pulmonary function tests, whereas previously they had not). However, such decisions are made behind closed doors and no record is kept of debates, or changes in policy within the Committee.

The use of medical review panels in other jurisdictions was reviewed by Barth in relation to asbestosis claims. He noted that in Quebec all workers in asbestos mines are given chest x-rays annually which are sent to the Commission des Accidents du Travail du Quebec where they are read by certified readers. If any characteristics of asbestosis are seen, the worker is examined by a three-person panel of respirologists, all of whom are present for parts of the examination establishing the diagnosis and assessing the impairment, and no screening of claims takes place once the x-rays have been read. The twelve experts, working in four teams of three physicians each, meet monthly to review their findings and assure some uniformity. Barth also noted that in contrast to Ontario, Quebec uses two pathologists working independently when a pathological decision is needed.

In contrast, Barth found that in British Columbia, no outside panel of medical experts is used in routine claims. Rather, a single staff doctor establishes the diagnosis and extent of impairment. Barth notes two reasons for this. First, British Columbia has far fewer asbestosis claims and secondly, B.C. is one of a minority of provinces to use outside medical review panels in appeals with the power to make binding determinations, thereby providing the claimant or his or her representatives with access to independent professionals to air any dissatisfaction with the medical judgement rendered.

8. Letters to claimants: Contributing to the sense of outrage felt by some injured workers toward the Board, is the inadequacy of WCB explanations of the facts upon which decisions in their cases are made. This point was discussed by Barth and has been noted in the course of the Weiler Inquiry as well. When a worker, keenly aware of his or her deteriorating health and increasingly disabled respiratory system, is merely informed that "your recent examination indicates that there is no evidence of an industrial chest disease" or even that "there is no change in your industrial chest condition" anger and distress often understandably emerge. (Copies of the form letters sent to claimants from the Claims Adjudication Branch were included in Barth's report.)

   It is difficult even for professionals working in the field of occupational medicine to appreciate some of the complexities involved in the Board's entitlement decisions without detailed explanation concerning the findings and the assumptions that were made. The worker certainly has the right to be properly informed of his or her health status and the issues behind the important decisions concerning his or her livelihood. The Board could do much to improve its public relations if greater effort was made to properly educate the worker as to the basis of the Board's decisions, whether or not they are favourable.

9. Permanent disability awards: Satisfaction or dissatisfaction with the Board cannot be measured by the numbers of allowed or rejected claims alone, particularly in reference to the pneumoconiosis claims. Workers are often dissatisfied if their percentage rating is much less than they perceive it should be. This situation is further complicated by the apparent indecisiveness of the Workers' Compensation Board on the question of impairment vs. disability. As was noted in the Evolution of Policy (see Appendix 1), Directive 2(d) of December 1955 stated that for assessing silicosis and pneumoconioses claims "percentage ratings are to be based on clinical findings, but where special consideration is considered warranted, the circumstances are to be specified." (Two subsequent directives further mixed the two concepts of impairment and disability: Directive 13 of June 1978 stated that "for interpretative purposes with respect to the Workmen's Compensation Act, disabilities should not only represent physical disability but also shall apply to a non-physically disabling condition which bars the employee from earning full earnings where the condition is connected with the employment and medically certified." It does, however, imply that medical assessment of impairment is still to be the guide in workers who are "physically disabled". In January 1980, Directive 5(c) incorporated the age factor into permanent disability awards to workers with job-related skin conditions. (The various policy decisions and assumptions that must be made in assessing disability attributable to workplace factors were enumerated in Section 1.) Cases illustrating the hardships and misunderstandings that are inherent in the current permanent disability rating system are discussed below.

   Many claims that are initially rejected are later reassessed, as the disease progresses, and subsequently allowed, and the percentage ratings themselves are frequently altered upon re-examination. Barth specifically looked at this issue in his study of asbestosis claims. He found that 30% of the individuals evaluated by the Advisory Committee on Occupational Chest Diseases who ultimately received compensation were initially rated as not impaired (0 rating) and that, in fact, a large proportion of claims from those initially rated at 0 impairment were eventually accepted by the Workers' Compensation Board. He also showed that a considerable number of persons in his study had been examined by the Advisory Committee on Occupational Chest Diseases on five or more occasions. Excluding the totally disabled and the claims evaluated in the last two or three years, he found that a few claimants had ratings changed four or five times since the initial assessment, but for many claimants the ratings were never modified despite several re-examinations. He noted that several of these may have died during the four-year period he reviewed and others may indeed have had conditions that were stabilized. His model applicant had one rating change over the four-year period. Barth also noted that persons with claims denied tended to be younger than those with claims accepted, as would be expected.

In principle, the process of continuous review by the Board is commendable. However, some people object to how it is put into practice. Cases illustrating the process of reassessment and the frustration associated with it are also presented below.

10. Rehabilitation: As was shown in Table IIIB-12, a disability pension may vary from 10% to 100%, but is not awarded where there is no impairment present, even if the disease is, in fact, recognized. This occurs frequently (or at least was occurring) in the situation of silicosis. Under such circumstances, the Board does not assume responsibility for rehabilitation or relocation except under certain circumstances such as those covered by the Board's special vocational rehabilitation program.

An assistance program was first devised in 1975 to offer rehabilitation to Elliot Lake miners. A similar program was devised in 1976 to assist asbestos workers. In 1977, it was expanded further to include other workers exposed to workplace hazards and became the Special Rehabilitation Assistance Program to remove workers from hazards of industrially-generated diseases. These programs demanded strict guidelines as to what constituted "pre-silicosis", "asbestos dust fiber effect" or "mineral dust effect" and were only available to workers "who are still subject to significant mineral dust exposure and who were of an age, education and motivation to be retrained or relocated in another field of endeavour." Three areas of controversy thus emerge: The first question is "what constitutes 'dust effect' and how is it distinguished from 'no effect' and from 'frank disease'? The second question is "what constitutes a hazardous environment?" and the third question is "on what basis are the other factors (i.e. age, education and motivation) determined?"

The Asbestos Assistance Program, thought of by many as a failure, was generally considered to have been politically motivated from the start. The Board's medical personnel had expressed concern regarding the ability to identify or define the asbestos fiber dust effect (AFDE) condition and were unhappy about the lack of evidence demonstrating that removing persons who have AFDE from low levels of exposure was medically beneficial. Conflict arose over what levels of asbestos exposure were required for eligibility. Moreover, many of the workers were older and unskilled and were hardly good candidates for retraining. An early retirement program would have been more suited to their needs perhaps. In any case, the whole endeavour proved fruitless. Ironically, while those without disability are given full benefits in the rehabilitation program, those too ill to qualify are left on their meagre 10% or 20% permanent disability awards. (Clearly, the only logical approach is to eliminate the hazards at the workplace so that no one is required to remain exposed to hazardous conditions, and to adequately compensate all those too ill to work, as well as those in the process of being retrained or relocated.)

11. The appeal process: As discussed in the first section, the appeal process has upheld virtually all initial decisions in disease cases. The most notable aspect of the appeal process is that again the dominant role is played by the Board's medical staff. Not only does the opinion of the Board doctor inevitably take precedence over the opinion of other doctors, but the Board doctors do not appear at the hearings and are therefore unavailable for cross-examination. Moreover, after the hearing, the proceedings are sent to the Board's medical staff for their comments with no formal provisions established to convey these comments to the other parties concerned, let alone offer the other physicians whose opinions are being challenged an opportunity for rebuttal. (Cases will be presented below which illustrate this point.)

    The recommendation of the White Paper with respect to the establishment of an independent (tripartite) appeal tribunal, as well as a medical review panel, has generally been well accepted in addressing this concern.

In summary, the identification of workers with pneumoconioses and the relationships between treating physicians who notify their patients about their potentially occupational respiratory diseases and the Board were discussed. The inconsistency of response by Board employed doctors to medical reports favourable to the worker as compared to those which were unfavourable has been identified as problematic. It was noted that neither the adjudicator, nor the Claims Review Branch, nor even the appeal personnel seem willing to significantly deviate from the recommendations of the Board's physicians. The lack of adequate explanation for decisions by the Advisory Committee particularly with respect to permanent disability ratings was also identified as problematic. This is compounded by the use of form letters rather than providing detailed counselling to the claimants as to the reasons for the decisions in their case. Frustration stemming from the inadequacy of the clinical impairment rating approach was acknowledged, as well as problems associated with the rehabilitation programs. The need for adequate appraisal of the workplace was mentioned, although is of much less concern with regard to the pneumoconioses as compared to the non-specific respiratory diseases to be discussed below. Criticism of the appeal process was also addressed.

Case examples will be presented to illustrate the points mentioned. The problem of "confounding factors" and policy in this regard are major topics of the next section.

2.3 *Case Presentations - Disability Assessment in the Presence of Confounding Factors and Eligibility for Rehabilitation*

Preamble: Although the pneumoconioses consist of many diseases aside from silicosis and asbestosis, specific review of pneumoconioses for this inquiry focused mainly on claims recorded in the silicosis index. No attempt was made to duplicate the work of Peter Barth who extensively reviewed asbestosis claims. A dozen or two asbestosis claims, however, were specifically reviewed at the suggestion of injured workers and labour organizations in their presentations to the Weiler Inquiry. Several of these cases have been included in the discussions below. As the intention of this study is to examine problematic areas in the process of claims adjudication, it should not be assumed that the cases presented below are typical; rather they have been specifically chosen to illustrate certain contentious issues identified in the course of this study.

As was shown in Table IIIB-10, there were 16 claims from the sample survey of 150 silicosis claims that went to the appeal process. These included 12 initially rejected (in 11 of which the rejection was confirmed and 1 in which the appeal resulted in the claim's allowance) and 4 allowed claims (which were contested by the employer with respect to cost charging). It was found that of the 12 rejections, all but one was rejected on the basis of the respiratory impairment being due to some condition other than silicosis. Generally, the issue contested by the claimant in these cases was not whether or not silicosis was present, but whether the worker's bronchitis, asthma, emphysema or even, lung cancer was work-related. In this sense, these rejections, although truly rejections, did not really belong in the silicosis index at all. (With this in mind, the statistics are much more in line with what would be expected: as silicosis is a prescribed disease under the Act once the diagnosis is established, the entitlement is all but inevitable.) Moreover, it reflects the observation made by a senior Board physician who specializes in respiratory disease claims, that "the problem of silicosis in Ontario is not silicosis but chronic obstructive lung disease." (In other words, many workers exposed to silica dust have chronic obstructive lung disease, and it is this disease, rather than silicosis, which accounts for much of their disability.) The one claim that was rejected on the basis of insufficient impairment (i.e. diagnosed as a "pre-silicotic") was a claim that was eventually allowed at a rating of 10% permanent disability, probably less due to the appeal process than due to the progression of the disease as discussed in Part A of this chapter.

Some of the rejected cases that went to appeal will be presented in the discussion of the work-relatedness of chronic obstructive lung diseases below (see Section 3.3.2). The cases presented below have been chosen to illustrate the hardships caused by the current system of disability assessment and eligibility for rehabilitation, particularly in the presence of other medical conditions. Themes that run through all the case presentations here, as well as in the following section, include the role of the attending physician, the extent of medical controversy itself, and the irrelevant factors that often crop up in the reasons for the decision.

Case 1 - Claim 39 This claim was instituted in June 1978 for a 56-year-old miner who had been exposed to silica dust for 33 years in various mines in the Timmins area. His family doctor, noting that an x-ray taken three years previously showed adverse changes consistent with silica exposure, had told him that he had mild silicosis and should avoid heavy manual work and dust. The claimant had noted shortness of breath and sharp chest pains on exertion which he related to work, and therefore requested assistance under the Special Rehabilitation Assistance Program to leave the exposure employment. However, the employer did not have any surface work available, so he remained underground until he finally stopped work in May 1979 due to the deterioration of his chest disability. (This was almost one year after his initial request.)

In September 1979 he was examined by a respirologist from the Advisory Committee on Occupational Chest Diseases who described pinpoint nodulation on his x-ray as representing silicosis, but noted that his pulmonary function tests showed only minor obstruction to airflow which could be due to cigarette smoking or to irritant fumes of any sort. He concluded that the claimant should be able to perform all normal activities although heavy manual work might cause some shortness of breath, which he felt was unlikely to be related to silicosis in any case. The request was therefore denied.

The claimant was re-examined by the Advisory Committee in June 1980. It was noted that he had quit smoking three years previously but had a persistent cough with a slight amount of sputum, as well as shortness of breath and fatigue. His pulmonary function tests again showed moderate obstruction, some slight restrictive defect, but normal diffusing capacity. His chest x-ray showed no significant changes from previous reports. The Committee, therefore, concluded that the claimant had Code 4 silica dust effects with moderate obstruction and restrictive ventilatory defect and apparent marked shortness of breath. They felt, however, that there was not enough radiological evidence to warrant a diagnosis of silicosis and hence, they recommended rejection of the claim and re-examination in two years' time.

The rejection was confirmed by the Claims Review Branch in May 1981, and an appeal followed. The solicitor for the employer agreed that the claimant was clearly disabled, but was unable to estimate if the disability was compensable or not. Following the hearing the Appeal Board directed that the claimant be re-examined by the Advisory Committee as soon as possible rather than waiting the two-year period. The Advisory Committee report was submitted in October 1981 and this time stated that the x-ray now showed signs of early silicosis. A disability award of 10% was granted and re-examination again scheduled for two years' time. (The results of the pulmonary function tests in 1978 and 1981 are shown in Appendix 2.)

Commentary: This 54-year old miner, as well as his own doctor, strongly felt that continuing at his job would be ill-advised for health reasons. The lack of suitable alternative work is perhaps the major issue underlying this case. Presumably this claimant would benefit considerably from the kind of WCB reform recommended in the government's 1981 White Paper.

Case 2 - Claim 40 This silicosis claim was for a 62-year-old miner, whose employment history was as follows: From 1939 to 1944 he worked in a dusty smelter, from 1944 to 1960 he worked underground in Ontario; he then spent two years in Saskatchewan underground; from 1967 to 1970 he worked as a cage tender at an Ontario mine, followed by a year as a surface worker at the same mine until he finally stopped work in 1971.

The worker presented with shortness of breath on exertion and a productive cough. It was noted that he had quit smoking in 1971. His pulmonary function tests showed severe airway obstruction (VC 3.54, 87% predicted; FEV_1 1.03, 33% predicted; FEV_1/VC 29%; $FEV_1 25\text{-}75$ 13% predicted, RV 4.15 l, 23% predicted; gas transfer 29%). His chest x-ray was noted to have sparse dispersion of nodular shadows toward the upper and middle zones, as well as transluscency of the lower fields and flattened diaphragms. The diagnosis was that he was disabled due to obstructive lung disease, but that the chest x-ray warranted a diagnosis of silicosis. His disability due to silicosis was estimated to be 25% and he was scheduled for re-examination in one year.

In July 1981, the Advisory Committee confirmed the diagnosis of silicosis as well as chronic obstructive lung disease and probably panlobular emphysema. His disability rating was increased to 50% and he was scheduled for re-examination in two years. While it was noted that this man's total disability was definitely severe, the fact that part of it was due to chronic obstructive lung disease prevented a higher rating. The employer contested being charged with the claim as the claimant had only one year exposure underground there. However, its request for relief was denied.

Commentary: This case epitomizes the problem of quantifying the work-related component in an individual with severe respiratory disability. In light of the material presented in Part A of this chapter, the 50% figure is clearly rather arbitrary.

Case 3 - Claim 41 In 1977, a claim was submitted for a 61-year-old asbestos worker who had worked for a manufacturer of asbestos products for 25 1/2 years. He had been diagnosed by his family physician as having chronic obstructive lung disease and active rheumatoid arthritis two years previously, and by the time of examination in conjuction with his WCB claim, the internist noted that he had been unable to work for approximately one year due to ill health. His pulmonary function tests showed slight airway obstruction with normal diffusing capacity and his chest x-ray showed pleural fibrosis, costophrenic bunting and linear fibrosis. The physician concluded that it was unclear whether or not the claimant had frank asbestosis, but there certainly were asbestos effects visible on the pleura. The claimant was examined by the Advisory Committee on Occupational Chest Diseases in August 1977 who concluded that although he had asbestos effects on the pleura bilaterally as well as a suggestion of pleural plaques on the surface of the right diaphram, he had no pulmonary asbestosis. They pointed out that he had a myocardial infarction in 1970, osteo-arthritis of ten years' duration, and rheumatoid arthritis which was grossly limiting his activity. They noted that although he had stopped smoking ten years earlier, he was mildly short of breath on exertion and had cough and sputum. No disability pension was recommended.

In August 1979, the Advisory Committee noted that the chest x-ray now did show slight pulmonary fibrosis consistent with asbestosis, and awarded the claimant a 10% disability pension. They noted that his pulmonary function tests showed only a slight obstructive ventilatory defect. More changes were noted in a subsequent examination by the Committee in 1980, but the 10% PD rating was re-affirmed.

An appeal hearing followed in which a brief was presented with medical documents including an x-ray report stating that the worker had asbestosis since 1973. The claimant's representative contended that the claimant was off work due to asbestosis, not arthritis which he felt was a "red-herring" in the case. He discussed the distinction between impairment and disability, quoting from several noted medical publications. He pointed out that x-rays are not an important evaluation of impairment. He contended that symptomatology, lifestyle and lack of earning capacity should also be considered and finally he objected to the mention of the other non-compensable disabilities in the medical reports by the Advisory Committee. The claimant's family doctor also stated that the patient was clearly more than 10% disabled and that his illness did indeed restrict him to the home. The internist also confirmed that the patient's shortness of breath was incapacitating and his pension should consequently be increased. The Appeal Board recommended that the claimant be reassessed earlier than the one year for which he was previously scheduled. In May 1981, he was again examined by the Advisory Committee and the 10% permanent disability award again confirmed.

Commentary: Quantifying the occupational component of a worker's disability is here again shown to be problematic. Questions about the entitlement of claimants to wage-loss benefits as recommended in the government's 1981 White Paper would continue to be controversial.

Case 4 - Claim 42 In January 1971, a claim for asbestosis was filed for a 48-year-old former employee of an asbestos manufacturer. In 1969 he had left work due to shortness of breath and joint pain. On examination by the Advisory Committee in 1971, it was noted that his x-ray showed insufficient changes to warrant a pension for asbestosis. In June 1973, he was re-examined by the Advisory Committee and a diagnosis of mixed dust pneumoconiosis was made, entitling the claimant to a 40% permanent disability award. He was re-examined in 1974, 1976, 1978 and 1979. No changes were noted by the Advisory Committee on any of these visits. It was noted, however, that he had spent a significant amount of time in hospital during this time and although his arthritis had been in remission since 1973 or 1974, his respiratory impairment had worsened. By this time he was requiring oxygen at home; he was very disabled and was hardly able to go outside. The oxygen he required was deemed compensable by the Board but no increase in the 40% PD award was recommended.

At the appeal hearing, the claimant's representative questioned the reliability of the tests performed by the Advisory Committee stating that he felt that it was unusual that in 1973 a 40% disability was awarded, but two years earlier the claimant had been deemed to have 0% disability. He submitted medical evidence that this disease was usually progressive in nature. He contended that there was little doubt that the claimant was 100% disabled due to his lung condition and that this was indeed recognized by the Board in its decision to allow compensation for the oxygen.

The Appeal Board noted that the chest x-ray showed minimal asbestosis, that his pulmonary function tests were basically unchanged over three years, that the non-compensable factors such as the arthritis contributed to the overall disability, that acceptance of payment for a mode of therapy does not necessarily reflect the degree of disability, and that in March 1981, the Advisory Committee did not find that the claimant required the oxygen. Consequently, in September 1981 the Appeal Board denied the claim.

Following the appeal, the claimant's representative sent a letter to the Board opposing the Appeal Board's practice of submitting appeal hearing transcripts to a senior Board physician for his comments. He contended that these views should have been presented at the hearing where they would have been open to cross-examination. He took issue with many of the observations made by this individual, specifically he objected to the contention that the assessment of 40% may have been in excess of the actual pulmonary impairment. The representative pointed out that the Board physician who reviewed the case never questioned the 40% assessment at the time. He also pointed out that the pulmonary function test results had continued to deteriorate despite the fact that the other medical problems disappeared. He noted that the Board doctor had contradicted himself by objecting to the observation that assessing disability is difficult in the presence of a number of serious medical conditions, in that he himself had earlier made the same observation. He also objected to the fact that despite never having seen the claimant, the Board physician attributed the use of oxygen to medical mismanagement: "Family doctors frequently prescribe medication of doubtful value....creating a psychic need for oxygen." He pointed out that the Occupational Health Clinic at St. Michael's Hospital did indeed find the patient mildly hypoxic, justifying the use of supplied oxygen. He summarized that no other condition was responsible for the claimant's need for oxygen and that this was recognized by the Workers' Compensation Board in accepting the cost. It was clear that the claimant was unable to carry on gainful employment and therefore, he strongly objected to the Board's refual to consider the claimant 100% disabled due to asbestosis.

Commentary: Providing yet another example of the difficulty in quantifying the occupational component of disability especially amidst divergent medical assessments, this case illustrates procedural and attitudinal characteristics at the Board which have frustrated claimants and their representatives.

Case 5 - Claim 43 This was a claim for a 54-year-old asbestos worker. In March 1977, the Advisory Committee awarded him a 10% permanent disability award for asbestosis, also observing that the claimant had chronic obstructive lung disease and chronic anxiety neurosis. He was seen again by the Advisory Committee in 1978 who noted that the claimant had been experiencing pain in the right side of his chest and had ceased to work. The x-ray at that time showed linear fibrosis bilaterally and pleural thickening. As this was not a change from previous chest films, the Advisory Committee reaffirmed the 10% permanent disability award. In 1979, the Advisory Committee again noted that the patient had asthma, obstructive airway disease, as well as chronic anxiety neurosis with phobic features; as the x-ray was still unchanged the 10% permanent disability was reaffirmed.

An appeal ensued in which medical documentation was presented, including a respirologist's report stating that "the history and findings are certainly consistent with pulmonary asbestosis". Again the union quoted from various medical articles indicating that pulmonary function tests do not always equate with the degree of disability and that neither x-rays nor pulmonary function tests reflect disability of the individual as a whole. It was stressed that he could not work due to his chest problem which required him to see his family doctor weekly. Again the Appeal Board requested the opinion of one of its senior physiians who specialized in chest disease and subsequently concluded that the claimant quit work on the advice of the union and not his doctor, that the x-ray and pulmonary function tests were the only way to assess disability, that the claimant did not try to complete the pulmonary function tests and that his main problem was asthma which was not related to asbestos exposure. The 10% disability was therefore reaffirmed and it was also noted that the patient was ineligible for special rehabilitation assistance.

Commentary: This case is included merely to emphasize that the problems outlined above are not restricted to isolated claims.

Case 6 - Claim 44 This 54-year-old miner with over 30 years' exposure to silica submitted a claim for silicosis in October 1978. It was noted that he had shortness of breath on exertion and an occasional cough but that he was never a heavy smoker and had actually quit smoking four years previously. His pulmonary function tests showed moderate chronic obstructive lung disease (VC, 92% predicted; FEV_1/FVC, 57% predicted). His chest x-rays showed increased markings in both lungs and diffuse small nodular shadows in both upper and middle lung fields. A diagnosis of early silicosis with moderate chronic obstructive lung disease was made and the functional disability due to the silicosis was estimated as 20%. He was scheduled for re-examination in two years' time.

In August 1979, both the claimant's family doctor and respiratory specialist confirmed the diagnosis of silicosis and recommended that he be placed in a surface job. The claimant had been told five years previously, when silicosis was first diagnosed, that he would be placed on the surface to avoid greater exposure, however, the transfer had never taken place. Meanwhile, he was increasingly short of breath even on slight exertion, he had a continuous cough and he felt strongly about getting out of exposure as his doctors had recommended. In September 1979 the Advisory Committee agreed to assess him sooner than scheduled, and he was re-examined the following February, instead of October. The Advisory Committee reported, however, that his pulmonary function tests were unchanged and that there was no radiological progression. They recommended that he remain as classified with a disability of 20% and be re-examined again in a year's time.

In November 1980, the claimant wrote to the Board stating that he had left underground work due to a knee operation and the recommendations of his doctor, but that there was a lot of difference in earnings and he therefore wanted a higher pension to enable him to meet his expenses. He was consequently re-examined in January 1980 by the Advisory Committee, however, the previous permanent disability assessment was reiterated. It was recommended, however, that he be referred to rehabilitation for re-evaluation. (Nothing further was noted in his file since January 1981.)

Commentary: In this case the issue was less related to the level of impairment as to the eligibility for rehabilitation. As was seen in the first case presented, medical opinion strongly dictated that this worker should not remain on his job, yet the mechanisms to facilitate this matter did not seem to function swiftly or smoothly.

Discussion:

The central controversy in the first five cases revolved around the percentage of disability award which the claimant was receiving for his recognized pneumoconiosis. In all the cases, the issue was made difficult by the presence of other medical conditions; specifically, obstructive lung disease. It was never stated if any component of the chronic obstructive lung disease was deemed compensable or if its enhancement effect upon the pneumoconiotic process was taken into consideration in the assessment. The very mention of the non-compensable disease was found objectionable by representatives of the claimants. In several of these cases controversy focused on the eligibility for rehabilitation; in others, the workers considered themselves to be totally disabled and requested compensation on this basis. In all cases, there was sharp divergence of opinion among medical professionals, with the adjudicator often favouring the view of a Board doctor rather than the claimant's. It was noted that Board medical personnel are not available for questioning at appeal hearings. Furthermore, their medical opinions are seldom successfully overruled. These issues have all been alluded to above and will be discussed again in the closing chapter with respect to possible solutions.

Case 2 introduced the dissatisfaction that exists with respect to cost charging. It is the policy of the Board to charge the cost of pneumoconiosis claims to the last employer where the worker had been exposed to the causative substance. This procedure is followed even if the worker may have been exposed in many workplaces and might even have been employed longer at one of these other than the last employer, as was the case in this claim. Barth noted that in some instances the Board had applied the claim's cost to all employers in that industry's classification. The Second Injury and Enhancement Fund, developed so as not to discourage the employment of workers with some type of pre-existing impairment, is widely used in Ontario, as in other jurisdictions. However it is not applicable to respiratory diseases as it is not desirable to encourage the employment of workers with respiratory disease in other environments which may aggravate their condition.

3. The Adjudication of Chronic Obstructive Lung Disease Claims

3.1 *Occupational Asthma*

3.1.1 *Policy on the Adjudication of Asthma Claims*

Each year the Ontario Workers' Compensation Board receives two or three dozen claims that are considered truly occupational asthma. The acceptance of these claims is not taken lightly as they often apply to young workers (in their early 20s) and have potentially far-reaching implications.

As was discussed in Part A of this chapter, an asthmatic component may co-exist with other chronic obstructive lung diseases complicating the adjudication of the latter. Its presence in an individual with pneumoconiosis also confounds the assessment as was illustrated by some of the cases presented above. It is not known how many respiratory claims are reported to the Board in which the principal diagnosis is finally determined to be asthma.

In the assessment of occupational asthma per se, the policy of the Board has been fairly well-formulated. In contrast, the adjudication of claims for other respiratory conditions in which it is found that there is a significant asthmatic component is not clear-cut. In Case #5 above, a worker with silicosis was found to have moderately severe asthma. His asthma was deemed non-compensable and his total disability award remained at 10% despite significant respiratory disease.

In 1962 the Board directed that a committee be formed to study certain aspects of allergic diseases in industry. The Committee's conclusions as submitted and approved by the Board, are shown in Appendix 3. With respect to heredity and age, factors acknowledged as influencing the propensity to develop allergic disease, it was stipulated that neither should warrant limiting entitlement under the Act. In the presence of an occupational, as well as a non-occupational asthmatic condition, the following was recommended: "Although an episode of allergic disease precipitated by one allergen may make an individual more susceptible to a similar illness provoked by unrelated allergens, such episodes are to be considered causally related only if the earlier condition is still active at the time of onset of the subsequent illness." This was applicable both to the situation where the initial asthmatic condition was occupational as well as in cases where it had originated non-occupationally. For example, the fact that a worker had asthma previously would be deemed irrelevant if the episode in question was felt to be occupational in origin. The committee specified, however, that where known prior non-occupational allergic disease is reactivated by a related allergen in the employment, entitlement is terminated with recovery from the acute episode. Where recovery does not occur from this acute episode, a permanent disability award could be considered. When a worker does not show improvement within six months after removal from exposure to the offending allergen the claim would be referred for assessment, and when it is felt that maximum recovery has been achieved the claim is brought to settlement. Once the compensable episode is stabilized and the permanent disability award made it is concluded that no further entitlement to medical aid should occur.

Due to apparent difficulties in applying the 1962 Board order, a revision was undertaken in 1963 and again in 1965 to streamline the procedure and implicitly alter the policy in reference to the pre-existence of non-occupational asthma. (See Appendix 3.) Allergic respiratory disease claims are presently referred for adjudication to two senior Board physicians one of whom specializes in occupational diseases claims and the other specializes in WCB claims for respiratory disease. Where a worker shows no improvement within three months of removal from exposure, the claimant is reassessed regarding continuation of entitlement and "to determine if any portion of the employee's continuing disability is attributable to a condition or conditions not included in his entitlement under the Act." A permanent disability award is considered when the compensable episode is stabilized and at least six months have elapsed from removal from exposure or last exacerbation. All permanent disability ratings are established by permanent disability consultants in consultation with Chest Disease Services. Medical staff in that department also determine the nature and extent of continuing medical aid.

Two distinct situations exist:

1. In primary asthma due to occupation which results in some pulmonary impairment, the worker may be retrained, depending on age, education and motivation. He or she is entitled to receive temporary benefits as well as medical aid and is reassessed every year or two initially regarding entitlement and the possibility of receiving a permanent disability pension. When relocated in the new job, a wage loss differential is paid.

2. In occupationally-induced asthma or asthma in a worker known to have asthma previously, even if quiescent at the time of the occupational episode, compensation will be paid for the acute phase only. Eligibility for retraining and a wage differential would be contingent on proving that the exposure imposed an additional impairment. (In this case the company would be eligible for relief through the Second Injury Enhancement Fund.)

The majority of asthma claims in the workers' compensation system, apparently do not involve permanent disability awards, since most workers are left without impairment. Six months or more later, once full recovery has occurred, if asthma develops again the claim is not automatically accepted.

Several difficulties encountered in the adjudication of occupational asthma had been identified by a senior Board medical consultant: "We have found there are only a few centres in the Province equipped to satisfactorily establish a diagnosis of occupational asthma, and I think it fair to say that in a substantial number of claims accepted by the Board, true cause or effect is never established, and often the suspect agent is never known or verified." The lack of certainty referred to was based on the premise that a definitive diagnosis necessitates a bronchial challenge test.

> There are instances in our experience where the temporal relationship between work exposure and onset of the classical symptoms of asthma were ideal, where there was no known previous medical history of

> relevance, but where bronchial provocation tests prove to be negative. (We nevertheless recommended to the Board that these claims be accepted.) We could only argue that the substances used in the challenges possibly did not contain the offending irritant or antigen and rarely do we ask for second challenge using another sample.

One cannot help but question why a bronchial provocation test was done in the first place, if indeed the temporal relationships were present, the symptoms classic and there was no competing explanation for the asthma. This would tend to lead to the assumption that had the temporal relationship and symptoms not been classic and had there been a pre-existing asthmatic tendency, a negative test albeit unreliable may well have militated against the allowance of the claim. It is clear that the improved availability of facilities for bronchial challenge testing would indeed help with diagnosis in a situation where the agent in question is known but the symptomotology of the individual was questionable. Nonetheless, it seems that there is a preoccupation with this supposed degree of certainty attributed to bronchial challenge testing which is unwarranted especially in the situation of mixed exposures and is time consuming, difficult and even dangerous. In fairness however, as a Board-employed physician points out, the Board "may accept a claim without a bronchial provocation test if the temporal relationships are right and particularly, the suspect causal agent involved is well known for its propensity to sensitize, for example, isocyanates and detergent enzymes."

An even greater problem identified is that while the investigation is proceeding, the individual involved may not be working. According to the same physician, "The Compensation Act does not allow for compensation to be given before the claim is finally accepted. While we may authorize whatever medical investigations may be required to evaluate the merit of the claim we cannot pay temporary benefits to the individual involved, if during these ongoing investigations he must stay off work. Ideally, on many occasions the patient should be removed from work for a period of several weeks to sort out the relationships involved." As it seems generally acknowledged that the removal of workers from exposure may not only be necessary from a treatment point of view, but also from a diagnostic point of view, one wonders why this apparent obstacle persists. It, in fact, is inconsistent with the entitlement to benefits during the period of definitive diagnosis which is allowed for chronic obstructive lung disease claims due to sulphur dioxide exposure and for emphysema due to alpha-1-antitrypsin deficiency, as will be discussed below. Moreover, it has been observed that the Appeal Board has occasionally granted total temporary benefits for a period of diagnostic observation. In fact, the Act neither specifically allows nor does it prohibit, paying benefits under these circumstances.

Besides the lack of bronchial challenge facilities and the reluctance of the Board to provide benefits to workers who are under medical investigation, further problems in this area have been identified. The Regional Chest Allergy Unit has noted that while frequently their representative is given permission to visit a workplace and evaluate the job, it seems only to be possible where there is no company physician. Almost invariably where there is a works doctor, the permission is not granted. (The law does not prevent a company from refusing a private physician entry to its workplace.) While the Board can ask the Occupational Health Branch of the Ministry of Labour to investigate and submit a report to the Board with respect to the job, the substances used and the state of the ventilation system, the report may not supply the investigating physician with what is wanted, and may even be misleading. (Cases illustrating these problems will be presented below.)

Another impediment which was identified is the reluctance of some companies to reveal the constituents of a particular process - apparently for reasons of trade secrecy. The identification of a product and its constituents can, however, be demanded by the Ministry of Labour.

Yet a further problem, perhaps the most serious of all, is the not-infrequent situation in which a worker is "let go" for filing a claim, while the Board investigation is neither able to identify a known pulmonary sensitizer in the workplace, nor does it find evidence of pre-existing asthma. According to one senior Board physician: "We find ourselves never able to confirm anything in the claim, but we are left with the impression and the feeling that the asthma would not likely be caused by that particular workplace. Without bronchial provocation, and in the absence of a known sensitizer, we would hope at least to have some evidence relating to the non-specific airway reactivity response to histamine. We would attempt to get that individual back to the workplace and by the use of the peak flow meter in conjunction with a histamine challenge test, we might be convinced that cause and effect is demonstrated. We presume that if the non-specific airways reactivity increases following renewed exposure, and only recovers slowly, the stimulus is an occupational one." In this case, entitlement would follow. However, if the workplace is indeed devoid of respiratory hazards and is unlikely to cause or exacerbate an asthmatic condition, there seems to be no justification at all for dismissing the worker. (Of course, there is also no justification for maintaining a hazardous workplace.) In any case, the phenomenon of dismissing workers for filing compensation claims is a serious problem which must be dealt with very strictly. Unfortunately workers - especially those from non-union shops - have stated that it is very difficult to prove that a particular dismissal was indeed related to the filing of a WCB claim and they are therefore intimidated from doing so. Worse, as will be illustrated by Case 9 below, a worker may be discharged "for health reasons" with the Compensation Board assuming no responsibility for relocation.

Thus, the adjudication of asthma claims is straightforward if the sensitizer is known, if the symptoms are classic and if there is no pre-existing condition. (Case 4 below illustrates this situation.)

However, more often than not, the allergen is not as yet recognized and the lack of full co-operation by the company coupled with the reluctance of the Workers' Compensation Board to provide workers with benefits during a period of necessary investigation makes the adjudication of these claims difficult. Moreover, often the worker is found to have some predisposition to asthma. This not only compounds the decision of whether or not the episode in question has an occupational basis, but even if the occupational exacerbation is acknowledged, entitlement to benefits is restricted to the acute episode unless the claimant can prove why the Board should accept responsibility for relocation or ongoing benefits.

Finally, the issue whereby the asthmatic condition effects the adjudication of claims for other respiratory diseases deserves mention. Some cases of this nature have been discussed, others will be discussed in the following subsection.

3.1.2 *Case Presentations - Occupational vs. Non-Occupational Asthma and Other Issues Concerning Benefit Entitlement*

Case 1 - Claim 45 This 54-year old miner and smelter worker filed a claim for "severe asthma bronchial condition". He worked in a nickel smelter for 38 years including a minimum of 52 to 156 days in the sintering plant in which he was exposed to a lot of dust and sulphur dioxide. He was also exposed to very dusty conditions in the mill and various gases in the smelter. The claim was denied and appealed. After the appeal hearing, the record was submitted for review to a senior Board physician who specializes in respiratory disease claims; he noted that the claimant had a "history of asthma sensitivity" and had developed nasal polyps years earlier. He also noted that there were no specific inhalants encountered at the workplace which prevented the claimant from working and that after a year out of exposure he had not improved. He felt that this was a classical case of adult-onset asthma of the intrinsic variety and concluded that the claimant's asthma did not arise out of employment.

Commentary: Case 1 has been chosen as a first case to present in this section as it illustrates the difficulties the Board faces in the adjudication of claims. The Board physician who reviewed the case clearly stated the Board's policy and its rationale for rejecting entitlement in a letter to the claimant's union representative: "Because a person develops asthma or chronic airways disease while working does not automatically mean that there is a causal association. If the individual shows no sign of improvement while outside his job or no ... deterioration while on the job, if the industrial environment has not been previously recognized as causing an increase in prevalence in chronic lung disease and that there is a ready explanation for the asthma, chronic bronchitis or emphysema, then the claim probably would not be acceptable. In this case, we have a clear inherited predisposition to develop asthma shown by his asthma sensitivity and development of nasal polyps many years previously." Nonetheless, as will be shown in Case 4 in Section 3.4.5, occasionally cases of this nature are allowed and the dividing line is quite vague and relies upon subjective judgement.

Case 2 - Claim 46 This claim for occupational asthma was filed by a 30-year old woman who was employed in a government office for 4 years. In September 1980, a heavy smoker moved into her poorly ventilated office. During the winter, the claimant had considerable problems with her allergies, but during the month of July, when the heavy smoker was out of the office for five weeks, the claimant noted that she was much more comfortable. After August 1st, when the smoker returned, the claimant noted increasing shortness of breath, sore throat and coughing, so she laid off work for a week. The Board also noted that since there had been some control of ventilation, the claimant has experienced no significant trouble at work. A WCB physician who specializes in industrial diseases, reviewed the claim and noted that the claimant was obviously an atopic person and sensitive to molds. She also observed that in August when the claimant was seen by the Board's physician there had been no change in her pulmonary function tests. Commenting that August is a month of heavy pollen, she doubted that the smoking was a significant factor in her lay-off in August and she referred the case to another Board physician - this one a specialist in respiratory disease claims for comment. The second doctor noted that the investigator confirmed how smoky the office gets and that the claimant had no problems prior to the entry of the smoker, and felt that the benefit of the doubt should be given to the claimant in this case. He recommended accepting responsibility for the week in question with the understanding that the Board would not recognize any permanent damage.

Commentary: Case 2 illustrates that differences of opinion do occur among Board doctors. The allowance of this claim was deemed due to exacerbation of a previous condition, although there was no pre-existing disability noted. (i.e. her likely predisposition to allergy, or atopy). This is also a major issue in Cases 7 and 9 below. The adjudication of claims in the "atopic" individual is certainly a very difficult area especially in cases where no real previous disease existed prior to the occupational encounter. This will be illustrated further below.

Case 3 - Claim 47: A claim was filed for an acute exacerbation of asthma for a labourer who worked as a mover. On the day of onset of severe wheezing and shortness of breath, he had to work outdoors in cold rainy weather. The diagnosis was an acute respiratory infection superimposed on bronchial asthma. The Board medical consultant who reviewed the case did not consider that the exposure described would be of adequate consequence to be of significance and the claim was denied.

Commentary: In contrast to Case 2, the exacerbation principle was not deemed applicable here, although it is certainly questionable why the Board doctor felt that the cold wet weather was irrelevant.

Case 4 - Claim 48: This 41-year old worker who had been employed in the steel industry for 18 years, filed a claim for aggravation of asthma due to work exposure to dust, smoke and oil mists. The claimant noted that in winter the ventilation was inadequate and he had complained to the employer about the dust and dirty conditions. In 1979 he had been told that he had asthma but subsequently was told that he had "a bronchial condition". The claim was consequently originally filed for "chronic obstructive lung disease" and rejected; it was later resubmitted as a claim for "aggravation of asthma". It was noted that the claimant had reduced his cigarette smoking from about 20 cigarettes per day to three or four daily and he did not classify himself as a heavy smoker. The company representative at the appeal hearing pointed out that there was much on file on the worker's asthmatic problem. He indicated heavy smoking and drinking were contributory factors and that no one else in the area had similar problems. He contested the accusation that the ventilation was not adequate. Furthermore, he mentioned that "Schedule 3 of the Workmen's Compensation Act shows no indication that asthma is currently recognized by the Board". A senior Board physician who specializes in chest diseases, reviewed the case and noted that the only improvement occurred when the claimant reduced his smoking and started inhalant medication and that he never had to stop work because of asthma. "The progression since 1967 is no worse than one would expect in a heavy smoker with atopy, allergic rhinitis and asthma. In other words, we need not explain the course of the disease by postulating the presence of an outside factor." The appeal was denied.

Commentary: A major reason for the rejection of this case was that the claimant improved on medication and reduction of cigarette smoking. The Board doctor failed to acknowledge that asthmatics indeed improve on treatment by definition and therefore the improvement on treatment should not be a major reason to infer lack of work-relatedness. (In fact, it is for this very reason that respirologists have been requesting that the Board allow workers total temporary benefits during a trial of removal from exposure at a stage when medication may be avoided and/or re-introducing exposure once medication is no longer needed, as a means of establishing the diagnosis of work-relatedness.) Interestingly, the claimant had felt that although his respiratory disease was rejected under one set of guidelines, it may be allowed on submission under a different category. Although this did not happen in this case consideration under all possible clauses, policies or guidelines should be automatic.

Case 5 - Claim 49: This 62-year old machinist who worked for 17 years for his company doing lathe and drill press work, filed a claim stating that his breathing was much worse at work. His asthma had begun in February 1980, with airway obstruction documented by decreased peak flow rates which he attributed to exposure to oil. It was noted that his chest problems had been initiated by shortness of breath and acute pneumonia five years previously, followed by frequent infective episodes, and that he had smoked until three years previously. The report of the plant investigation by the Occupational Health Branch stated that exposure to oil did, indeed, exist, but that it was not a sufficiently high concentration to constitute a serious health hazard.

In September 1980, the claimant had an acute asthmatic episode which started mildly at work on Friday after the cutting oil fumes were again present. He stayed off work Monday, and Tuesday when he returned to work, he became increasingly breathless and had to be hospitalized for nine days. Treatment was begun and it was noted by his respirologist and documented by clinical evaluation and peak flow recordings, that the claimant was well when he was off work and worse when he was back on the job. Actually it was noted that he was fine when he returned to work until cutting oils were used and then within two or three days he would become severely asthmatic again. The respirologist at the Chest Allergy Clinic stated: "I agree that he should stay off work for the time being unless he can be assigned to a part of the factory where he is not exposed to these fumes... The history seems pretty clear-cut and I am convinced that he does, in fact, have a problem in relation to his work." It was later noted that he had increased bronchial reactivity to histamine even after about two months off work, suggesting severe asthma which would have been expected to have been less following such a long cessation of exposure, but that possibly the sensitivity had caused a permanent increase in reactivity. It was pointed out that although there was no clear pattern of response, the claimant indeed had shown gradual deterioration since his return to work. As the exposure to cutting oils was intermittent, it was difficult to assess. A bronchial challenge test had been done which showed no evidence of sensitivity. The respirologist noted that the work-relatedness remained inconclusive but that the claimant would be wise to avoid exposure.

The Board doctors decided that they could not accept the diagnosis of occupational asthma, but could entertain accepting limited responsibility for aggravation of a non-occupational asthma. They felt that the Board's responsibility should be limited to well-defined periods of exacerbation of asthma which occurred specifically due to the cutting oils, and that no permanent disability responsibility should be accepted by the Board. The final diagnosis was intrinsic asthma associated with severe non-specific bronchial hyper-reactivity. It was noted that the claimant also had hypertension, heart disease, and recent transient ischemic attacks (small strokes). It was concluded that his layoff from May to July 1981 was really not attributable to asthma and therefore wage loss benefits could not be allowed either for this period or for the numerous trips the claimant made from St. Catharines to the Regional Chest Allergy Centre in Hamilton for investigation of his asthma. His travel expenses and medical expenses were, however, paid.

Commentary: This worker was on and off work for the greater part of a year while his case was being adjudicated. Although he did improve on being removed from exposure and became worse after returning to work, his negative bronchial challenge test suggested that the work-related aspect of his asthma was not through sensitization, but by non-specific aggravation. He was ultimately not only denied a permanent disability award, but was also unable to receive benefits for his lost time, much of which was necessary in the diagnostic work-up leading to the ultimate decision about the work-relatedness of his condition. The bind workers find themselves in, is that if they do not get better while off work (for example, Case 1) the condition will likely be deemed not work-related; if they improve on medication but remain on the job, it leads to the conclusion that the work-related component is insignificant (for example Case 4); if they take time off to see if they get better while off work without treatment or, worse, if they take time off to have definitive diagnostic tests done (for example, Case 5) not only do they risk losing their claims as a result, but the lost time is at their own expense. Furthermore, as will be shown in Case 9 below, they risk losing their jobs and even jeopardize future job possibilities. These factors could be strong influences in discouraging workers from filing claims.

Case 6 - Claim 50: This worker attributed his chronic obstructive lung disease, diagnosed as bronchial asthma together with pulmonary fibrosis, to his occupation as a welder over a period of 30 years. His shortness of breath began 15 years previously but he was never treated until 1978 when he had an upper respiratory tract infection. He had no hay fever or other allergies. He never smoked more than five or six cigarettes per day and had no past history or family history of asthma. He had not worked since January, 1981 stating that he had been declared totally disabled for employment due to his pulmonary problems. Over the years he had performed many different types of welding. Prior to 1979 he used no protector and stated that the area was poorly ventilated.

The claim was denied and at the appeal hearing the claimant's representatives provided medical articles supporting the work-relatedness of his condition, including case reports of pulmonary fibrosis in arc welders. As well, he provided evidence of management's response to the union's grievances over fumes in the workplace, as well as their attempts to improve the conditions. Physicians of the Occupational Health Clinical Unit at St. Michael's Hospital confirmed the diagnosis of chronic obstructive lung disease and asthma, attributing the problem, at least in part, to employment exposure. Another specialist provided the diagnosis of asthmatic bronchitis aggravated by cigarettes, as well as diffuse pulmonary infiltrates characteristic of welder's lung, but stated that he did not think that the occupational factor was all that significant and certainly it was not the only factor in the respiratory impairment. Yet another specialist concluded that the claimant had moderately severe asthma, making it unlikely for him to be able to undertake any active, manual occupation. He stated that the asthma was probably exacerbated by his occupation, although he could not state with certainty that the occupation had indeed caused the asthma in the first place. He did, however, conclude that the fibrosis of the lung was probably of occupational origin. A senior Board physician who reviewed the case, stated that the asthma had not been shown to be occupational in origin and in fact, was triggered by an upper respiratory tract infection. He noted that following this episode, the condition deteriorated even after the claimant began working in a well-ventilated area. Also he stated that there was no evidence that the diagnosis of possible "welder's lung" bore a causal relationship to the claimant's exposure over years of employment. He stated that the inability to work was solely due to asthma in any case. The appeal was thus denied.

Commentary: This case illustrates the controversy within the medical profession regarding the work-relatedness of respiratory disease. Despite the fact that many physicians, including an occupational medicine specialist, supported the work-relatedness of the claim, the Board doctor felt differently (and even implied that the "welder's lung" component of respiratory impairment, although minimal, was not related to welding!). The readiness to attribute the disease to an upper respiratory tract infection is certainly questionable. (Many welders have filed claims with the WCB for chronic obstructive lung disease, as will be illustrated further in Section 3.4.5 and much has been written in the scientific literature suggesting a possible relationship.)

Case 7 - Claim 51: This 33-year old worker filed a claim for occupational asthma. A plant investigation was requested but the claimant noted that a few days prior to the actual inspection by the Occupational Health Branch, a massive clean-up was instituted. Moreover, he noted that during the visit, the paint booth site of the exposure in question, was not in operation. The occupational respirologist at the Regional Allergy Centre who was also the attending physician in this case, stated that the Occupational Health Branch had not provided sufficient information. An air quality assessment survey was not conducted. At the appeal, heard February 6, 1980, the Appeal Adjudicator requested that air sampling be done. On May 5, 1980, a senior Board physician who specializes in occupational disease claims was reviewing the case and he indicated that the plant had been visited, but that sampling had not yet been performed. On June 11 and July 8, further follow-up requests were made. On July 25, 1980, the Board doctor received a call from the Ministry of Labour that there was no need to survey for air pollutants. However, as the worker had written to the Chairman of the Workers' Compensation Board and received a verbal commitment that the sampling would be done, the Board doctor called the Occupational Health Branch on August 5 and was subsequently advised that the air sampling would indeed be carried out. On September 3, the results were received - seven months after the specific request by the Appeal Adjudicator.

Meanwhile, the Board doctor had noted that the claimant had an upper respiratory tract infection for some months prior to commencing his employment. However, it was conceded that a sensitized pulmonary tree could react unfavourably in any environment including possibly this one. As a result, it was recommended that the Board accept limited responsibility based on aggravation, recognizing that while the diagnosis of occupational asthma could not be confirmed, the working environment was such as to aggravate a pre-existing chest condition. Benefits were paid from January 20 to February 19, 1980 at which time it was felt that the claimant was able to return to employment. As the claimant had not sustained any permanent disability as a result of his exposure, the services of the Board's Vocational Rehabilitation Branch were denied. After the hearing, the claimant's supporters, including his attending respirologist, stressed that there was no documented pre-existing chest condition before the onset of asthma and that the causal factor was indeed the workplace. The respirologist noted that cleanliness of the work environment does not mean that an individual cannot be sensitized to limited exposure. He pointed out that the claimant had become highly sensitized which would effect his employment in his trade as a machine fitter, and that retraining and rehabilitation facilities of the Board should be made available to him. It was also pointed out that retraining in this case would avoid the necessity for compensation on a permanent basis and would greatly decrease the likelihood of a new claim for aggravation of asthma in the new work environment.

Commentary: Case 7 was chosen mainly to illustrate the dissatisfaction with the role of the Occupational Health Branch in carrying out the industrial hygiene assessment in conjunction with the claim. The respirologist of the Regional Allergy Chest Unit had suggested that better communication between the attending physician and the industrial hygiene team would possibly alleviate the problem, or, alternatively, the Board may approve the carrying out of this sort of investigation by certified outside laboratories. The other reason for presenting this case was to illustrate the hardships inflicted upon the claimant as a result of considering him "atopic". The alleged predisposition to asthma was based on conjecture and, in fact, strongly opposed by the respirologist. As shown, the decision has far-reaching implications beyond the denial of a permanent disability award since it also entails the denial of rehabilitation assistance. This case illustrates that even when a claim is allowed, the claimant may end up "losing".

Case 8 - Claim 52: This claim for occupational asthma was filed by a 26-year old worker with no allergies, asthma or family history of asthma. He had been hired in September 1977 after working for the same company from 1972 to 1975, both times in the scarfing and foaming departments. He had had his first pulmonary function test in 1977 or 1978 and, as another one in June 1979 showed deterioration, the company doctor suggested that he leave the department. He had shortness of breath, chest pain, and also had a rash on his arms. It was noted that there were a number of other workers in the same department that had to be removed from exposure. As there was no pulmonary function impairment demonstrable due to the isocyanate exposure in May 1981, it was recommended by the Board that he receive no award at the time, but be reviewed on further request or on evidence of deterioration in pulmonary function.

His case was accordingly reviewed in August 1981 at which time it was felt that a 10% permanent disability award was in order as well as a temporary wage loss supplement as the claimant, clearly having become sensitized to isocyanates, was unable to return to his regular work. The Claims Review Branch concluded that even though his disability was not demonstrable in the absence of exposure to isocyanates, nevertheless, it did constitute a permanent restriction upon his ability to work. The company appealed this decision. However, the Board physician who reviewed the case, pointed out that not only was the claimant forever barred from working with isocyanates, but that sensitization from such a chemical often resulted in hypersensitivity of the bronchi to other substances or in other circumstances often of a non-specific nature including exertion-induced bronchial spasm. As to the question of whether or not the claimant would have found himself in a position to be laid-off even if he had not become sensitized, the Board doctor pointed out that this was primarily a claims problem and not a medical one, the sensitization clearly effecting his re-employment leverage being the real restriction. The company's request was thus denied, the P.D. award provided and the temporary supplement extended for another three months while the Rehabilitation Branch helped relocate him.

Commentary: Case 8 was a clear contrast to Case 7 and shows how the situation can be handled in a more just manner - albeit at the expense of the employer. Compensation on the basis of a condition's effect on the individual's re-employment possibilities is of considerable importance, especially in these hard economic times.

Case 9 - Claim 53: This 41-year old woman who worked at an electrical appliance factory for 12 years, developed cough, shortness of breath and wheezing on February 9, 1981 after being at work five to six hours. She was worse within an hour or two of leaving work. It was noted that she had no fever and it was felt that she had either a viral illness or a "reaction to something", however, she was put on antibiotics anyway.

Returning to work on March 2, her cough and shortness of breath became more severe within two hours or so, so she went to the Emergency Room where she was treated with a bronchial dilator. She remained off until April 20. On her first day back, the symptoms began to recur after four hours and became progressively worse over the next two days. She remained off work and her symptoms continued to improve. It was noted that she worked on an assembly line approximately 20 feet from an area where isocyanates were used and about 20 feet in the opposite direction from an area where soldering was carried out. Apparently the ventilation was very poor.

Her past medical history revealed that she had had rhinitis and nasal polyps in 1978 and 1980, which was thought to be due to inhaled allergens and she had received allergy injections. Her nasal symptoms were no worse at the time of her chest symptoms and she had never had chest symptoms previously. Skin prick tests were also negative. It was noted that she had moderately severe airflow obstruction which was hardly improved by bronchial dilators.

Her respirologist, felt that her cough and shortness of breath were clearly induced by exposure to materials at work and probably as a result of asthma. He concluded that the diagnosis of asthma was probably previously missed due to the predominance of cough with no wheeze. He thought that the sensitizing agent was probably isocyanates, although it may have been materials in the soldering process. He felt that "she should not return to work in the same area because of the severity of her symptoms, which could be life threatening, and because of their chronicity which could induce irreversible changes". He noted that the firm would not transfer her, therefore she would have to leave the firm despite the fact that she would have to do this before the Workers' Compensation Board had approved her claim.

After being away from work for three to five months (at her own expense) she continued to have mild to moderate airflow obstruction and mild non-specific bronchial reactivity. The respirologist hypothesized that her continuing irreversible airflow obstruction and bronchial hyper-reactivity on the one hand could be sequelae of occupational asthma, but on the other hand could be the result of another disease which had developed coincidentally. However, he noted in this connection that she had not had previous chest symptoms and had not smoked, so the latter was unlikely. He also noted that she was depressed due to being dismissed by the company in such an offhand manner and had been looking for another job, preferably in cleaner surroundings. He asked that the Rehabilitation Branch help her to accomplish this.

The Board notified the Occupational Health Branch requesting a workplace assessment. Meanwhile, the company sent a letter to the Board pointing out that the initial diagnosis was bronchitis and that the patient was not totally disabled. They stated that she also had other conditions, for example, anemia, and that the diagnosis of work-relatedness was speculative as she still had mild asthma even though off work. They mentioned that patch tests for fibreglass inhalants were negative and stated:

> We are of the opinion that the claim was not work-related based on (1) the vague terminology used by doctors and the lack of scientific evidence to verify that a product from her work environment caused her attack; (2) it was proven that she had had no allergy to inhalants; (3) she had a history of breathing difficulties; and (4) regulation 118/8 insists that if the disease is not scheduled it has to be shown that the disease is caused by employment and is not due to any other cause.

A Board physician who specialized in industrial disease cases was consulted and she noted that the Occupational Health Branch reported that isocyanate exposure was most minimal. She stated that according to the reports, the claimant had an infective episode in February 1981 and that she had a history of allergic rhinitis and hay fever indicating that she was a person with a respiratory atopy. This Board doctor therefore, concluded that the asthma was not significantly influenced by her work and referred the claim to another WCB physician who specialized in claims for respiratory disease for comments. In discussing this claim with the attending respirologist and another specialist in respiratory diseases, the Board doctor concluded that possibly the claim had enough merit to warrant some allowance.

By June 16, the respirologist consulted by the claimant, felt that she was capable of returning to work and as there was still a discrepancy as to whether or not her asthma was work-related, returning to work would give her exposure one more time and therefore offer an objective way to confirm whether or not her surroundings caused her asthma. As the employer would not allow her to return the doctor felt that the claimant should be entitled to rehabilitation in order that alternative work could be found. A Board physician agreed that any acceptance of this claim would involve full work incapacity benefits from April to June 1981 and that entitlement should also be extended for rehabilitation services. However, in view of the fact that the claimant was capable of returning to work June 16, the Board concluded that benefits beyond this stage should be denied. Benefits were thus paid from April to June 16, 1981 only.

Shortly afterwards, a letter was sent from the claimant's respirologist to one of the Board physicians involved in the case summarizing some of his concerns:

> I am concerned about the potential misinterpretations and attitudes reflected in the letter to me dated September 15, 1981 (from the first Board physician who reviewed the case) specifically about (1) the relevance of prior rhinitis, atopy and chest disease, (2) the recognition of what is likely to be of occupational asthma induced sensitization versus what is coincidental asthma aggravated by exposure to irritants at work, and (3) the obstructive attitude of employers which prevents complete investigation. The claimant had prior rhinitis but no prior atopy (as defined as an increased tendency to form IgE antibodies to common allergens). She had no previous chest disease, was a non-smoker and no alpha-l-antitrypsin deficiency to predispose her to chronic obstructive lung disease. In occupations where potential hazards exist, I think it should be the responsibility of the employer to demonstrate that prior chest disease was or was not present. Her clinical features are highly suggestive of occupational asthma caused by sensitization. Complete investigation was obstructed by her employer who had not let her return to work because they felt, against our advice, that it would be dangerous to her health. Their actions suggest that they were concerned that something at work was causing her chest disease and that they did not want this to be identified. I

> strongly feel that this approach should not be encouraged and I wonder how this can be conveyed to them or other employers who adopt this attitude. I am very concerned in such cases of which claimant number 52 (Case 7) was another, that the eventual benefit of doubt comes down in favour of the employers rather than the patient. The patient is the loser because even if compensation is paid for a period, they often find it difficult to obtain further work either because of their age, or because the previous employer gives them a rotten reference.

Commentary: This case epitomizes the problems in the adjudication of occupational asthma. First, as pointed out by the claimant's specialist consultant, it illustrates the Board doctors' difficulty in sorting out the role of sensitization versus aggravation, and the preoccupation with atopy. The readiness to accept that an upper respiratory tract infection triggered the event was also unwarranted. (It is well-known that the prescription of antibiotics, especially in an emergency room setting, does not necessarily prove that an infection was present, let alone that it was causative.)

Secondly, this case illustrates the potentially misleading nature of the information supplied by the Occupational Health Branch in investigating the workplace. Also, as was mentioned, even a small amount of isocyanate could stimulate an attack once sensitization has occurred and hence the cleanliness of the workplace should not be cited as a reason for rejection.

Thirdly, the company not only played an obstructive role in the investigation of the claim as was also pointed out, but the reasons given by the company as to why the Board should reject the claim were most interesting. They mentioned the fact that the claimant had other medical conditions, the fact that she had been previously diagnosed as having bronchitis not asthma (erroneously) and that she had a negative response to a patch test (irrelevant). They also clearly pointed out that the onus of proof is on the claimant and consequently were able to contend that proof of work-relatedness was not adequately established. Such is the situation facing workers with possible occupationally related asthma.

3.2 *Policy on the Adjudication of Pulmonary Emphysema due to Alpha-l-Antitrypsin Deficiency*

The main issue with respect to the adjudication of claims from workers with emphysema has been the relative roles of hereditary, smoking and occupational factors. In January 1977, a senior member of the Board's medical staff reviewed the literature on the subject of the hereditary factor, specifically the role of alpha-l-antitrypsin deficiency in chronic obstructive lung disease.

A genetic predisposition was first suggested in 1963. It is now known that there are more than 20 genetic deficiency combination pairs in the general population which, the Board physician concluded, may result in premature and/or abnormally severe chronic obstructive lung disease and emphysema. These genetic deficiencies result in varying abnormally low levels of an enzyme produced by the liver, called alpha-l-antitrypsin, which is needed to neutralize excess digestive substances produced by certain white blood cells. These latter substances, called the proteases, normally function to preserve the integrity of the alveolar-capillary complex by aiding in the removal of foreign particles. (See Part A of this chapter.) If there is a deficiency in alpha-l-antitrypsin, there may be an excess of proteas (trypsin) in the lung, which in its vigour to digest bacteria, cellular debris and inhaled particulates, will proceed to digest or destroy the alveolar walls resulting in characteristic emphysema. In the intermediate type, the affected individual has one abnormal gene and one normal gene (called heterozygous), while a severe deficiency is characterized by two abnormal genes (homozygous). Some authors have suggested that the risk of emphysema is inversely related to the level of antitrypsin in the serum under stress. For instance, in the homozygous state, 90% of the antitrypsin may be absent, while in the heterozygous, 40% is absent. In between are the various genetic combinations. The resting level of alpha-l-antitrypsin in the blood is not a reliable indicator of risk, since when activated, its level may increase 2 to 25 times as a result of various stresses. In deficiency, ability to increase the level of antitrypsin may not be present, although the absolute resting level may be normal.

The genetic deficiencies have been estimated by some authors to be present to some extent in up to 15% of the general population with a greater incidence among those with proven chronic obstructive lung disease and emphysema. It has been stated that possibly 30% of affected individuals have some genetic deficiency, while in those under age 50 with these conditions, up to 50% might have genetic deficiencies. Although the disease is basically the same whether or not an individual has a special predisposition to develop it, a senior physician employed by the WCB, reviewed the literature about the subject and concluded that the latter group seemed to develop the clinical signs and symptoms much earlier than those with no deficiency. Some evidence has suggested that a significant percentage of those affected do not have a history of chronic bronchitis preceding the first clinical signs, the emphysema is primarily confined to the lower lung fields and is panlobular rather than centrilobular.

As would be expected, studies show that smoking has a major influence on the manifestation of the condition. Some authors believe that only a minority of non-smoking heterozygotes seem to develop the disease, although it has been suggested that heterozygotes do have an increased risk and homozygotes have an almost 100% chance of developing emphysema. There is controversy not only about the actual percentage of non-smoking heterzygotes who develop the disease but also regarding whether or not the heterozygotes are more susceptible to the harmful effects of environmental hazards than those without the deficiency. As the Board doctor remarked: "It is difficult to avoid concluding that many occupational exposures will have the same potentiating and aggravating effects as those clearly demonstrated by smoking."

Over the next year additional information was sought and a set of guidelines was recommended. The Board felt that the principle of activation of a predisposition to environmental exposure should be applied to the situation of alpha-l-antitrypsin emphysema and consequently the

Board should accept some responsibility for the development of this disease in workers subject to certain hazardous exposures. The occupations specified included mining, firefighting, coke oven exposure, welding, smelting of base metals, foundry exposure, pulp and paper making, etc. Some of the most important substances involved were specified as nitrogen oxides, chlorine, ozone, sulphur dioxide and ammonia.

The literature reviewed by one physician employed by the Board led him to the observation that intermediate deficients i.e. people with the heterozygous state (mz gene pair - 1 normal and 1 deficient gene) who present with manifest premature disease usually have 15 or 20 years or more exposure prior to the onset of symptoms. Hence, it was recommended that a minimum exposure in any of the above environments be at least 15 years, particularly in the absence of smoking.

Because cigarette smoking clearly influences the frequency of emphysema, it was felt reasonable to apply a differential disability rating based on the genetic susceptibility and smoking history in each case. In homozygote (zz) smokers, the probability of occurrence of the disease is high, so it was felt reasonable to allow a maximum of 25% to the occupational factor. In homozygote (zz) non-smokers, the occupational factors would be of greater significance in terms of age of onset of the disease, although it was felt to likely be of little significance in the probability of the ultimate occurrence. Therefore, a maximum occupational relationship of 50% was proposed. In heterozygote (mz) smokers, the probability of developing the disease was concluded to be approximately a factor of 3 over an mz non-smoker hence it was recommended to assign the occupational factor a weight of 50%. In the mz non-smoker the probability of occurrence of the disease was felt to be small, hence it was recommended that an occupational factor of 100% was indicated. (See Appendix 4 for the complete guidelines.)

It would logically be expected that the smaller the genetic predisposition to develop the disease, the greater the role attributable to occupational factors when the disease does indeed occur. One would further assume that this logic would be extended to those without any known predisposition so that normal (mm) non-smokers who develop the disease in certain occupational settings would be entitled to a 100% award and normal (mm) smokers would be entitled to at least 50%. This extrapolation does not however occur and, in fact, those without the genetic predisposition seem to have no entitlement at all. A possible rationale for compensating those with a predisposition and not compensating those without it is to discourage companies from employing workers with this predisposition. Use of these tests for pre-employment screening was, in fact, alluded to several times in the background papers produced by the Board's consultant in conjunction with the recommendations. Regardless of the acceptability of this discriminatory practice, this policy of awarding benefits only to 'susceptible' workers is inconsistent with Board policy in all other areas.

With regard to age of onset, the senior physician who conducted the literature review found that 90% of disease in deficients appears before the age of 60 and therefore concluded that the first symptomotology must be present before 60 years of age if the claim is to be allowable.

As chronic bronchitis and asthma are not invariably present in those with emphysema due to alpha-l-antitrypsin deficiency, it was felt that it was necessary to distinguish these diseases from emphysema and a series of guidelines for differentiation was proposed. (See Table IIIB-14.)

As alpha-l-antitrypsin deficiency results in the diminished elastic recoil of the lungs leading to alteration in the ventilation-perfusion balance, it should not lead to an increased susceptibility to respiratory infections, hence it was felt that even in the presence of a confirmed genetic deficiency, acute respiratory infections should not be compensable in the absence of demonstrable impairment of ventilation. In the presence of impaired ventilation, an episode of lower respiratory tract infection, for example, pneumonia may be given serious consideration.

The Board physician doing preparatory study for the development of emphysema guidelines, recommended that total temporary benefits be paid when surgical intervention was warranted in a compensable case, during the definitive medical investigation preceding the confirmation of diagnosis, and during acute lower respiratory illnesses where indicated. Based on these recommendations, a series of guidelines was developed. Following discussion by the Board, some changes were made. For example, whereas in the first set, the "ex-smoker", defined as someone who had not smoked for ten years or more, was considered a non-smoker, in the revision this definition was eliminated and adjudication for an ex-smoker was to be based on cessation interval and onset of symptoms. This imposed a further potential restriction at the discretion of the Board. Further discussion at the corporate board resulted in slight wording changes and formulation of the guidelines as they stand today. (See Appendix 4.)

The most important issue identified in reviewing the formulation of these guidelines is the differential payment based on genetic predisposition and particularly the inconsistency of not allowing emphysema claims at all in those without predisposition. Although the Board does state that emphysema and chronic bronchitis are allowable according to the circumstances of exposure; case review and a look at the statistics have shown that the vast number of claims of this nature have not been allowed. The fact that blood tests may not be definitive in establishing the genetic predisposition and therefore, actual genetic testing is required, is also problematic in terms of the complexity and length of the time required to settle the claim. (See Case 2 below.)

Further issues identified relate to the inclusion of the various restrictions (age, years of exposure, etc.) based on very flimsy scientific data. The problem of restrictions incorporated into guidelines based on a somewhat arbitrary cutoff of probabilities was identified and discussed at great length with respect to cancer claims.

It is noticeable that the period of investigation to confirm a diagnosis is considered compensable in these cases in contrast to the situation discussed for asthma claims. This entitlement, however, has never been granted.

Finally, the lack of participation by an outside party either from the scientific community or from labour, management or the public at large in the development of this policy is also to be noted.

3.2.1 *Case Presentations*

As stated, the most obvious issue with respect to the adjudication of claims from workers who develop emphysema is the peculiar role allocated to the extent of genetic predisposition. Only four claims for workers with proven alpha-l-antitrypsin deficiency emphysema have so far been processed by the Board, of which three were allowed and one was rejected. Ironically, if two miners subjected to exactly the same conditions of exposure over the years both develop emphysema, and one has a genetic predisposition but the other does not, it is the worker with the predisposition who will be compensated. This anomolous situation is illustrated by the following cases.

Case 1 - Claim 54: This claim was established in June, 1981 for a welder of 30 years who was diagnosed as having bullous emphysema. He had progressive shortness of breath and cough, was a heavy smoker, and had a long history of diagnosed emphysema. His pulmonary function tests were consistent with fairly severe emphysema. His chest x-rays showed tremendous bullae bilaterally with extensive destruction of tissue also indicative of the disease. There was no question that this welder did indeed have emphysema, but the question posed by the Board was "is he susceptible?" An alpha-l-antitrypsin level was done which showed a value of 160 (the normal range is 200-400). In view of what were considered borderline results, arrangements were made for him to have genetic phenotyping done at Sick Children's Hospital in Toronto. Meanwhile, the claimant had suffered a stroke and has been unable to have the tests performed. The adjudicator noted that no further action would be taken in the interim.

Commentary: The major feature in this case was the fact that allowance or rejection of the claim rested entirely on the results of genetic testing - a curious situation indeed.

Case 2 - Claim 55: This 64-year old worker, employed as a cutter-grinder from 1955 to 1973, submitted a claim for emphysema. It was confirmed that he was a non-smoker and that his work exposed him to considerable metal dust from grinding. His alpha-l-globular level was assessed and it was found to be normal. A Board-employed physician who reviewed the case, noted that there was no temporal relationship between his increasingly severe asthmatic-like episodes and any particular phase of his work. He also pointed out that there were no studies showing a relationship between chronic obstructive lung disease or emphysema and grinding dust. He, therefore, concluded that this worker could be one of 5% who develop this disease without a history of smoking. Moreover, he commented that none of the claimant's physicians had discerned the cause as work-related and he therefore recommended rejection of the claim.

Commentary: If indeed there was no evidence to link chronic obstructive lung disease or emphysema to grinding dust, one would be led to question the relevance of testing the claimant's enzyme levels. It might have been thought that his being a non-smoker would have considerably strengthened the claim, but apparently this was not sufficient to shift the balance to the claimant's favour. The fact that none of the claimant's physicians attributed his disease to workplace factors is of questionable relevance for reasons discussed at length earlier in this report.

Case 3 - Claim 56: This 54-year old welder of about 20 years developed severe pulmonary disease diagnosed at that time as asthma and emphysema, which led him to apply for a permanent disability award in 1977. He apparently had been a cigarette smoker of less than one pack per week, but quit completely in 1976. During the investigation it was learned that the claimant had chronic bronchitis and emphysema as far back as 1951 when he had been hospitalized for an acute bout of bronchitis which was then followed by frequent upper respiratory tract infections. It was noted as well that he welded inside transformers, but that the other workers were not affected.

A Board physician who reviewed the claim concluded that workplace exposures had not been significant influences on this man's chest problem. He agreed that the onset of the chest condition had nothing to do with his work, however, surprisingly, he suggested that "as a vulnerable person he will be more likely to be affected by welding fumes". He therefore recommended that 20% of the disability be attributed to occupational causes. He commented that "this does not mean that I feel that in all cases of chronic lung disease and emphysema in welders that a cause and effect is established - it is the peculiarity or uniqueness of the chronology of events that precede the claim. Here the stainless steel welding, the presence of bronchial sensitivity and the hyper-vulnerability which might well have resulted from a sickness during the war all suggest the propensity to be injured."

The adjudicator initially presumed that the claim was to be allowed on the basis of aggravation of a pre-existing disability which would have entitled the company to relief from the Second Injury Enhancement Fund. However, on discussion with the manager of the Board's extended disability section, he concluded that this claim must be rejected:

> As the man was not a smelter worker exposed to sulphur dioxide the claim does not meet the guidelines for adjudication of claims for chronic obstructive lung disease. Secondly, we have not been able to establish a causal relationship between the exposure to welding fumes and his chronic lung disease. The picture is further clouded by the fact that the man had pre-existing chronic bronchitis and emphysema dating back to 1951.

The claim was therefore rejected.

Commentary: This was the only case encountered in which a Board doctor's recommendation to allow a claim was not heeded. The approach to this claim, truly on the basis of its own merits, was indeed impressive. Reference to the fact that this claim

Table IIIB-14

Guidelines in Differentiation of Stable Obstructive Airway Diseases

Test	Emphysema	Chronic Bronchitis	Asthma
Spirometry	Obstructive pattern with no bronchodilator improvement	Obstructive pattern may show some bronchodilator improvement	Obstructive pattern usually shows definite bronchodilator improvement
Sputum	Not helpful	Predominantly neutrophils	Predominantly neutrophils
Chest X-Ray	Helpful if evidence of bullous lesions or loss of peripheral vascular markings suggestive of presence of flattened diaphragms (hyperinflation)	Not helpful	May be helpful
Diffusing Capacity	Less than 50% of predicted; higher values do not exclude the presence of emphysema	Normal to moderately reduced	Normal in stable patients
Lung Compliance (Static)	Increased	Normal	Normal, may be increased during sustained hyper-inflation

Source: C. Stewart, Internal Memo Re: Guidelines for Adjudication of Emphysema Claims.

failed to meet established guidelines underlines the important role that guidelines play in the adjudication process.

Case 4 - Claim 57: This 58-year old miner submitted a claim for chest disability diagnosed as emphysema, bronchitis and asthma. He worked underground for a nickel mining company from 1941 to 1958 where he was apparently exposed to "dust, dampness and fumes from machine exhaust and blasting". After leaving that company in 1958 he worked for mines in Elliot Lake for a year or so, when he suffered a severe back injury and was off work for five years on disability. It was noted that he smoked approximately six cigarettes per day until 1959 when he quit. The claimant's representative cited documentation of acute pulmonary edema from exposure to blasting fumes and speculated on the chronic effects of this exposure on the pulmonary system. The company noted that the claimant did not have silicosis or other pneumoconiosis and there was no evidence of significant dust exposure. A Board doctor who reviewed the case did not consider this disability work-related as medical studies had not established that emphysema, chronic bronchitis or asthma are "peculiar to or characteristic of" the occupation of underground mining. The claim was, therefore, denied.

Commentary: In this case, alpha-1-antitrypsin levels were not ascertained. If a deficiency had been found, this claim would have been allowed. Part A reviewed the studies linking chronic bronchitis to underground mining. Clearly non-specific respiratory diseases such as emphysema, chronic bronchitis and asthma are not "peculiar or characteristic of" mining or other occupations for that matter. However, as discussed in Chapter I, Part B, this in itself has not prevented the Board from allowing other non-specific diseases on other occasions.

Case 5 - Claim 58: This 55-year old worker filed a claim for bronchial asthma and emphysema having been exposed to fumes from sludge or chemically treated human waste for 25 years. Although he mainly drove a truck, his job required him to remove the lid and put his face into manholes of treated sewage where he described exposure to "very strong fumes". He had been short of breath since 1970 and had been told that he was suffering from emphysema and bronchial asthma since 1972. He had apparently smoked half a pack of cigarettes per day for 35 years, but did not consider himself a heavy smoker and gradually reduced his smoking until he finally quit two years previously. The pulmonary function test results provided by his physician were consistent with significant airways obstruction and emphysema. A Board physician recommended that there was no information to suggest that the claimant was exposed to anything in the course of employment which would produce asthma and therefore recommended rejection.

Commentary: Here, too, the test for "susceptibility" was not performed, and without an industrial hygiene assessment it was not known if this case would have qualified under the guidelines.

Discussion:

The workers in all five of the above cases had evidence of emphysema. In Case 1, it is anticipated if the genetic testing proves that he is "susceptible", entitlement will follow. Ironically, in Case 2, in which the worker was exposed to working conditions that are included in the list of hazards to "susceptible" workers, the claim was denied because the worker did not prove his genetic susceptibility despite the fact that as a non-smoker, there was little other explanation for his disease offered. (The questionable accuracy of the blood test for "susceptibility" should also be kept in mind.) It is of interest to note that one of the factors cited as a reason for rejection by the Board's doctor was the fact that the claimant's physician had not contended that the work-relationship existed.

In Case 3, it was most surprising that the Board physician who reviewed the claim recommended allowing it since the vast majority of claims for chronic obstructive lung disease among welders are systematically rejected. (See cases below.) The rationale for recommending entitlement was the increased susceptibility of this worker to the hazards. Although it is most unusual for an adjudicator not to follow the recommendation of the Board's doctor, it was not surprising that this was the situation in this case.

Case 4 would also likely have been allowed had the worker been shown to have a genetic predisposition, as his occupation was also listed among those in the alpha-l-antitrypsin deficiency guidelines. Here, as in many cases reviewed, the alpha-l-antitrypsin tests were never performed. A senior Board physician questioned about the case, responded that the Board could not be expected to test everyone with emphysema who filed a claim. (Case 2 in Section 2.3, a totally disabled silicotic with emphysema receiving a 50% permanent disability award for his silicosis, might have been able to receive total disability benefits if he had proved his susceptibility.)

In Case 5, the worker's claim might not have been allowed even if he was able to prove susceptibility, as without an industrial hygiene survey, there was insufficient information to assess whether the exposure to the "strong fumes" would qualify as "prolonged exposure involving for example, chlorine, acid mist, ammonia, oxides of nitrogen, metallic fumes, sulphur dioxide and ozone." Neither was the alpha-l-antitrypsin deficiency test done, nor was an industrial hygiene investigation carried out.

The provision of a list of "industrial processes or occupational exposures which may be considered hazardous" is a step forward, since although it may not be inclusive, it certainly increases the likelihood of compensation being provided to those workers who develop emphysema due to prolonged exposure to the identified processes. However, the policy of allowing claims only by those proven susceptible is most curious indeed.

3.3 *Policy in the Adjudication of Chronic Obstructive Lung Disease Claims (Excluding Alpha-l-Antitrypsin Deficiency Emphysema)*

As mentioned, the only guidelines that exist with respect to the adjudication of claims for chronic obstructive lung disease, other than those just discussed for alpha-l-antitrypsin deficiency emphysema, are the guidelines that were developed to handle claims from workers exposed to sulphur dioxide and particulates.

As discussed at length in Section 3.2.2 of Part A of this chapter, in 1973 the Ministry of Labour conducted a study consisting of a questionnaire and limited pulmonary function tests among workers exposed for 10 or more years in a converter plant at Copper Cliff. The study was completed in September 1973 and updated in 1976. Although the study suggested that there was an increase in chronic obstructive lung disease due to prolonged exposure to sulphur dioxide particulates in the smelter industry, the results were felt to be inconclusive. Inquiries, notably from the Steelworkers Union, prompted a review of the data by a senior Board physician. In conjunction with the Ministry of Health, the Board decided that further studies would be justified.

Meanwhile the Royal Commission on the Health and Safety of Workers in Mines came forward with its report. It recommended "that workers in reduction plants who have been exposed for 20 years or longer to sulphur dioxide at levels approaching the current threshold limit values, and to associated dust and fumes, and who exhibit the clinical diagnosis of chronic bronchitis and impaired pulmonary function as identified by objective tests, be considered for compensation at up to a maximum of 20% disability."

No further studies were mounted by the Ministry of Health nor subsequently by the Ministry of Labour, although the then newly created Occupational Health and Safety Division was well aware of the request that further studies be carried out. In November 1978, the Ministry of Labour decided that the study would be returned to the Board to determine further action. Various meetings followed in which the Board requested that the Occupational Health and Safety Division of the Ministry provide research funding for this endeavour. In August 1979 the Assistant Deputy Minister confirmed that no activity related to an investigation of chronic obstructive lung diseases was going on in his division, nor was any activity contemplated in the foreseeable future, nor were any funds identified or allocated for such studies.

It was estimated that a formal study attempting to establish a clear relationship between sulphur dioxide exposure and chronic obstructive lung disease would require a study time of two to three years, including the time for analysis, and would be extremely expensive, estimated at approximately $500,000. Thus, on the basis of the Ministry of Health data and the review of the world literature, and in considering the recommendations of the Ham Commission, the Board agreed that certain cases of chronic obstructive lung disease would be accepted on a limited liability basis in appreciation of the multi-causal factors involved.

A set of guidelines was therefore developed and approved by the Board in September 1979. These specified that claims for chronic obstructive lung disease with impairment would be favourably considered "when all the following circumstances apply regarding exposure intensity, latency (inception), and cessation factor..." (See Appendix 5 for complete guidelines.) It was specifically stated that primary asthma without chronic obstructive lung disease would not be acceptable. The smoking history was to be taken into consideration when establishing percentage of permanent disability: smokers would be entitled to 20% of the assessed permanent disability, ex-smokers, 30%, and non-smokers, 40%. (Thus, if a non-smoker was rated at 60% disability, he or she would receive a 24% P.D. award; if he or she smoked, the P.D. award would be 12%.) Total temporary disability benefits would be paid for periods of disability due to acute lower respiratory tract illness, subject to appropriate medical evaluation, only in established claims for permanent disability, except "if required during the definitive medical investigation in order to confirm the diagnosis, when so ordered by the Board". With respect to emphysema and bronchitis in general, the 1973 Session Book states: "The Board takes into consideration the exposure, toxicity and irritability of industrial materials and other factors peculiar to the industrial process, trade or occupation, and entitlement may be granted on the evidence. Where emphysema and bronchitis are complications of such conditions as silicosis and pneumoconiosis, they are recognized as such and the increased disability they produce is provided for." The statistics of allowed claims for emphysema and bronchitis indicate that, in fact, few claims result in a favourable adjudication, and, as shown in the case presentations in Section 2.3, an increased award due to chronic obstructive lung disease (COLD) is not borne out in practice. (This is because COLD is generally not a complication of pneumoconiosis, but a separate disease as explained in Part A of this chapter.)

The issues raised here therefore include the following. First, the jurisdictional issue as to who is responsible for providing the funds and conducting the needed research was at least contributory to the long delay in developing the guidelines, (at least six years). This was similar to the situation described with respect to the foundry guidelines discussed in Chapter II.

Secondly, the action that was decided upon in the absence of definitive evidence was interesting in that it represented, in a sense, a compromise solution - allowing the claims, but placing considerable restrictions upon eligibility. Moreover, the percentages assigned to permanent disability awards, based on the recommendations of the Ham Commission, are themselves somewhat arbitrary. Nonetheless, the facts show that despite the restrictions, many more workers are being compensated for their disease through the application of these guidelines than were being entitled to benefits when claims are adjudicted "on their own merit". This will be illustrated by some of the cases presented below.

3.3.1 Case Presentations

As stated, the only guidelines for chronic obstructive lung disease (other than alpha-1-antitrypsin deficiency emphysema) are those for chronic obstructive lung disease in cases of proven sulphur dioxide exposure. The manner in which these guidelines are applied is illustrated by the following cases:

The Application of the SO_2 Guidelines

Case 1 - Claim 59: This claim for chronic obstructive lung disease diagnosed as "chronic bronchial asthma with emphysema" was submitted for a refinery worker who had been directly exposed to sulphur dioxide for 24 years in the converter. He apparently had asthma since 1966, although it was noted that another physician said he had asthma since age 14. The claim was rejected in January 1978, but in September 1979, M.P.P. Elie Martel asked for a review of the claim in light of the new guidelines. On review, it was noted that the claimant indeed had severe pulmonary function abnormalities with no obvious asthmatic component and that he smoked 4 to 5 cigarettes per day: "He is now 100% disabled from pulmonary impairment. He was a smoker, although a mild one. Our guidelines clearly state that the maximum for a smoker would be 20% of the rateable disability - a non-smoker would be eligible for 40%. In this case, the smoking habit is somewhat unclear. I would recommend that we settle on a difference between 20% and 40% which is 30%." A 30% permanent disability award was thus granted.

Commentary: The fact that this claim, rejected prior to the formulation of the guidelines, was allowed after their formulation again attests to the importance of guidelines.

Case 2 - Claim 60: This 56-year old worker was exposed to sulphur dioxide in converters for 26 years. He developed marked airway obstruction with emphysema and finally went on pension in 1972. It was noted that he smoked until six or seven years prior to diagnosis. The Board doctor who reviewed the claim considered him virtually 100% disabled and therefore recommended a 20% disability award as specified under the guidelines.

Commentary: This case illustrates a straight-forward application of the guidelines. Presumably this worker would have been eligible for wage-loss benefits if the Government's proposed reform of Workers' Compensation contained in a 1981 White Paper, had been implemented.

Case 3 - Claim 61: This 56-year old worker who spent 32 years working in a smelter submitted a claim for his chest condition. The medical evidence was reviewed by a senior W.C.B. physician.

> This man's problem is primarily asthma although there may be a fixed element of airways obstruction also, i.e. a true asthmatic bronchitis...High gas concentration tended to irritate him, but this also occurred with cold weather...This is not as clear cut a case as we would like in order to estimate contributability to sulphur dioxide...He, however, has over 30 years in the worst area of the smelter for sulphur dioxide...He has been a heavy smoker and his present disability is no more than moderate. By stretching the guidelines I feel that we can at the maximum set an award of 15%...Normally I would specify that this has to be the maximum award for

responsibility that the Board should accept, but I would in this case be prepared to recommend an adjustment on reassessment in three years' time.

A 15% P.D. award was thus granted.

Commentary: Presumably the 15% figure was derived from estimating 20% of his assessed total disability. The reference to the "maximum responsibility that the Board would accept" likely relates to the Board policy that the asthma component is non-compensable. It does not seem that the aggravation that the work-place may have had on the asthmatic condition per se was taken into account in this award.

Case 4 - Claim 62: This worker, employed in a nickel smelter for 32 years, filed a claim for asthmatic bronchitis. A Board physician who specialized in chest disease claims reviewed the sulphur dioxide levels within the various areas of the smelter and found that the total length of exposure at greater than 5 parts per million was only 8 years 6 1/2 months. He concluded that there was insufficient exposure in excess of 5 p.p.m. for the 20-year requirement in the guidelines to be met. He also noted that the claimant had had no exposure since 1963 which exceeded the cessation interval of five years specified in the guidelines. He, therefore, recommended denying the claim. An appeal was launched in which the exposure data were reviewed by the Ministry of Labour's Occupational Health Branch. It was concluded that he, indeed, had 18 years' exposure exceeding 5 parts per million. Suggesting the benefit of the doubt, a Board doctor recommended that the claim be accepted. He assessed the disability at 25% to 40%, and as the claimant was a smoker, the Board's responsibility would reflect 20% of this. A permanent disability award of 10% with no change was thus granted by the Appeal Board.

Commentary: It should be recalled from Part A of this chapter that individuals vary considerably in their response to dusts and fumes. Thus rigid adherence to guidelines, as illustrated by the initial rejection, may not be justifiable. The difficulty in ascertaining the true extent of past exposures is also illustrated in this case.

Case 5 - Claim 63: This worker claimed entitlement to total temporary benefits for a six-month period in which he was disabled due to bullous emphysema which required surgery. He worked for one nickel company from 1951 to 1964 and then for a second from 1964 until 1980, during which time he had been exposed to significant sulphur dioxide. It was noted that he had quit smoking by 1975. He claimed that his disability was either caused or aggravated by his work environment. The Board doctor who reviewed his claim, pointed out that the existence of the bullae had nothing to do with sulphur dioxide as emphysema is developmental or congenital in origin. He felt that the mild airway obstruction was likely due to the persistence of bullous change and noted that the worker smoked 30 cigarettes per day for 25 years. He concluded that the mild narrowing of airways could easily be explained on that basis and one need not postulate sulphur dioxide as a factor necessary to explain the degree of airways change. The appeal adjudicator noted that the claimant did indeed have the required 20 years' exposure to sulphur dioxide, however, accepting the opinion of the Board physician that the bullous emphysema was not caused or aggravated by sulphur dioxide, it was concluded that there was insufficient evidence to support a compensable pulmonary impairment. The claim was thus rejected.

Commentary: This case was rejected despite technically meeting the specifications of the guidelines. This illustrates that the guidelines are indeed rebuttable.

Case 6 - Claim 64: This claim was submitted for a 57-year old mechanic for his condition of chronic obstructive lung disease. He worked for a major nickel company for 37 years and the claimant was of the opinion that he was exposed to various gases including sulphur dioxide, because they were blown by the wind to the smelter area of the mill where he was employed. The union representative contended that because of the location of the mill and the smelter, he would have been exposed to a greater extent than the people who were actually working in the smelter itself. The company pointed out that this claim did not meet the guidelines and the Board doctor felt that the wind factor was insignificant. The claim was therefore rejected.

Commentary: Without a careful industrial hygiene appraisal it is difficult to evaluate the merit of this claim. However, the fact that it did not meet the guidelines, again, should not necessarily be a reason for rejection.

Discussion:

The existence of guidelines allows for greater entitlement than would be the case had they not been developed, as illustrated by Case 1 which was allowed only after the guidelines came into effect. However, percentage of entitlement is seriously limited as workers who are acknowledged to be 100% disabled in terms of their respiratory impairment are only entitled to 20% to 40% of the applicable disability benefits, according to their smoking status (Cases 1, 2, and 3). These levels fail to even keep pace with the permanent disability levels specified in the alpha-1-antitrypsin deficiency guidelines under which some of these claimants may possibly have qualified had they been tested. (Note that in Case 1 the claimant was a "mild smoker" and therefore was awarded 30% of his P.D. assessment, but in Case 2, although the claimant had stopped smoking for 6 or 7 years, he was still considered a smoker and awarded 20% of his P.D. assessment.)

Some flexibility was observed in the allowance of a claim held to be primarily asthma (Case 4), despite the fact that the guidelines specify that these claims may be rejected. As discussed in Part A of this chapter, the allowance of the claim is indeed warranted in view of the large overlap both in terms of etiology and clinical presentation of the diseases in the chronic obstructive lung disease group.

Claims were also rejected due to insufficient impairment (Case 5) and wrong location of employment (Case 6). Total temporary benefits were denied in Case 7 due to the congenital nature of the underlying impairment, rejecting an aggravating effect despite sufficient exposure. The question of sufficient exposure also was problematic in Case 4. The importance placed on the smoking history is evident as will be further illustrated in the next set of cases.

Smoking vs. Occupational Factors:

Case 1 - Claim 65: This 61-year old miner submitted a claim stating that he was off work since 1977 since he could not get a job because of his lung impairment. He worked approximately 30 years underground in hardrock mines including 12 years in Elliot Lake, known for its high dust exposure. He also spent a number of years welding. It was noted that he stopped smoking in 1974 and that his shortness of breath began in 1976. An x-ray report of 1978 was "suggestive of silicosis". A doctor in Thunder Bay saw the claimant in 1979 and stated that he had some silicosis based, first, on taking into account his underground mining exposure, secondly, the dry rales in both lung bases, thirdly, his pulmonary function tests which fit pulmonary fibrosis because of the abnormal diffusing capacity and finally, because the chest x-ray showed a reticulo-nodular pattern in both lung fields which was compatible with silicosis. In February 1980, another physician reported that he had chronic obstructive lung disease with pulmonary fibrosis confirmed by x-ray studies. He also pointed out that it was impossible to state whether silicosis played a predominant part in his respiratory impairment or not. The union presented various medical articles and hazard data sheets and pointed out that his work history was very conducive to the development of industrial lung disease.

The Advisory Committee on Occupational Chest Disease saw the claimant in February 1979 and in May 1980 and felt that there was no x-ray evidence of silicosis. They felt that the progression in the lower zone's haziness in the chest x-ray as compared to previous films was probably due to "pectoral soft tissue mass and possibly some bronchiectases". (Fat, muscle and collapse of some small airways.) The Adivsory Committee concluded that although the claimant had considerable underground exposure to silica, there was no x-ray evidence of silicosis and the chronic obstructive lung disease of the chronic bronchitic type "could well be due to his smoking habits", (The fact that the diffusing capacity was low was deemed also to fit that progression of events.) They, therefore, concluded that there was no significant work-relatedness and the claim was therefore rejected.

Commentary: Medical uncertainty regarding diagnosis was illustrated in this case. It is surprising that the many studies linking chronic bronchitis to underground mining outlined in Part A of this chapter were not alluded to by either the claimant's physician, the Board medical staff who reviewed the claim, or the ACOCD. In view of the 12 years of underground mining in Elliot Lake, it is particularly curious that a study which showed that dust exposure in Elliot Lake does indeed correlate with chronic bronchitis was not mentioned. Rather than asking if smoking could have accounted for this man's disability, perhaps the question that should have been posed here is whether working conditions could have been contributory.

Case 2 - Claim 66: This 65-year old miner claimed entitlement for chest disease diagnosed as chronic bronchitis. He had worked underground from 1947 to 1978. It was pointed out that he had smoked for 15 to 17 years, but was not a heavy smoker. He had earlier submitted claims for compensation for silicosis which were denied due to the lack of x-ray substantiation. In 1980 the shortness of breath was diagnosed as being related to obesity and chronic obstructive lung disease and his claim was again rejected. The family doctor who had supported the claim was irate and wrote:

> I am unable to understand how the conclusion is reached that Mr. R's chest difficulty is not of occupational cause. He is not and never has been a heavy smoker and he has a history of severe and prolonged dust exposure. There is no doubt that his obesity is somewhat contributory to his problem but the basic fact remains that this man has developed chronic obstructive lung disease during heavy dust exposure without being a heavy smoker. The conclusion is rather inescapable.

Nonetheless, the Appeal Board upheld the rejection stating that there was no evidence of chronic bronchitis as a result of employment.

Commentary: Again divergence of medical opinion was expressed, and again, the Board doctor's opinion prevailed.

Case 3 - Claim 67: This 48-year old worker submitted a claim for his chest condition. He had contacted pneumoconiosis while employed in Belgian coal mines for seven years prior to 1959. He then came to Canada and was employed in an incinerator from March 1968 to 1979. He was symptomatic for three or four years and had become short of breath even at rest. His pulmonary function tests showed mild airway obstruction. The worker claimed that his exposure in the incinerator had effected his condition. A work assessment was done and confirmed that indeed the claimant worked under very dusty conditions. Although respirators were available, their usage was not enforced. The Board concluded: "This man has mild obstruction quite consistent with his history of smoking up to two years ago and certainly there need be no other explanation for it. He does have pneumoconiosis but this is of Belgian origin. No Ontario occupational disease is present."

Commentary: Although it is commendable that a workplace assessment was carried out, one wonders if this was a futile exercise in that the Board's approach suggested that the claimant's respiratory impairment would be attributed to his smoking regardless.

Case 4 - Claim 68: This 40-year-old welder filed a claim for chronic bronchitis and emphysema. It was noted that he smoked one pack of cigarettes per day and also that he had a long history of exposure to welding fumes and dusty conditions. He commenced working in the mines in 1958 and after six months began welding which he did until he left in 1962. From 1963 to 1972, he did various jobs and then he began working on contract in the nickel industry where he claimed he was exposed to heavy sulphur concentrations and other gases. He was advised by his attending physician that he should not be employed where there would be exposure to welding fumes due to his chronic bronchitis and emphysema and by 1980 he was forced to seek help from the Canada Pension Plan. A senior Board physician assessed the claim and wrote that his symptoms could be explained by smoking alone. He felt that there was no evidence that the years of exposure to welding fumes were contributory.

Commentary: It seems that this man had been exposed to dust, welding fumes and SO_2. It is not at all clear why the Board doctor concluded that these exposures were non-contributory.

Case 5 - Claim 69: This claim for fatal benefits was filed by the widow of a 64 year old welder who died of a cardiac arrest in 1980 following a ventricular arrhythmia resulting from chronic obstructive lung disease and hypoxia. The deceased had worked as a welder from 1947 to 1948 and then continuously from 1960 to 1977 when his respiratory problems began. In December 1977 the claimant's respirologist had written to the Board that he had developed a progressive cough, breathlessness and bronchospasm which had gradually worsened over the preceding 6 months and had finally caused him to become so badly disabled that he was unable to work. Within a month he had become critically ill with acute bronchospasm, severely abnormal pulmonary function tests and a chest x-ray compatible with chronic bronchitis and emphysema. It was noted that the claimant had been working in a shop where up to 12 welders often worked at once, usually 3 or 4 working with stainless steel. The respirologist highlighted the noxious fumes produced in this procedure, which, he reported, caused all the welders to cough. He stated that the ventilation was poor and he referred to the scientific literature noting the link between welding and obstructive lung disease. He also noted that the claimant had stopped smoking 7 years prior to the onset of symptoms and was firmly of the opinion that "the main contributing factor in the cause of his respiratory problems (was) the air pollution to which he has been exposed at his job from 1960 to 1977." After accumulating all the medical records, a Board physician noted: "All in all it is a rather difficult case to analyze and while his work exposure no doubt has played a part in the initiation of the acute condition, its persistance out of exposure and the diffusion effect suggestive of some interstitial disease indicates the possibility of non-compensable changes..."

Although indicating his uncertainty with respect to the portion attributable to occupation, the Board physician estimated the disability at 70% and the disability claim was allowed, as was the fatal claim.

Commentary: The issue of fatal benefits will be examined at length in Chapter IV. The inclusion of this case in this discussion is to illustrate that, in contrast to the last case, the link between welding and chronic obstructive lung disease was indeed acknowledged here by the Board.

Case 6 - Claim 70: This worker was employed for 34 years at a porcelain company where he was exposed to fumes from hot presses, as well as the accumulation of silica dust in the area. He had become increasingly short of breath over the years and by 1980 had developed very severe airways obstruction. He did not have significant cough or sputum, nor emphysema, and there was no evidence of silicosis.

He was seen by an occupational respirologist who was also the Director of Occupational Health at McMaster University. He noted that although the claimant was a cigarette smoker for many years, there was reason to believe that the workplace may have contributed significantly. In a letter to the claimant he wrote:

> Although no clear distinction between cigarette smoking and dust exposure at work can be made, there is evidence that the effects of cigarette smoke are much more likely to result in emphysema. This is not present in your case and is evidence to support the idea that some factor other than cigarette smoking was the cause. .....There has been much dispute over the exact relationship between airborne dust measurements and the development of obstructive airway disease. That there is such a relationship is beyond dispute....I believe, in law, that you are entitled to receive the benefit of the doubt. Some doubt would be present unless there is documentary evidence concerning the gravimetric size selective dust concentrations to which you were exposed at work.

He also enclosed several articles.

A senior Board physician commented at great length on the letter. The points he stressed included the fact that the articles enclosed dealt with coal miners and therefore were not equally applicable to non-coal miners. He noted that in the British coal mine studies, the FEV_1 was only marginally reduced even after 30 to 40 years at the coal face and therefore was of no compensable significance. He quoted from Morgan "that cigarette smoking has a 5 to 10 times greater effect on ventilatory capacity as measured by FEV_1 than does coal dust." He noted that the claimant had severe reduction in his FEV_1 with a flow of only 0.8 litres per second and that "even if we do assume the results of studies on British coal miners should be applicable to employees of (the claimant's employer) adding another 200 to 300 cc to this man's FEV_1 would still leave him totally disabled, i.e. is of no clinical significance."

The plant was investigated and it was written that "most individuals did not feel the work environment was a dusty area, nor could they recall any other workers similarly affected."

Citing the above reasons and that "other doctors did not state that his obstructive airways disease was work-related," a Board physician recommended rejecting the claim.

Commentary: This case is a fitting one to end the discussion of the adjudication of claims for occupational respiratory disease at the Ontario Workers' Compensation Board. Not only were the comments of a world-reknowned occupational respirologist somewhat flippantly dismissed, but even the literature provided by the claimant was only selectively reported upon in the memo to the adjudicator. The most recent article enclosed was a letter from one of the authors involved in the British Coal Mine studies (Jacobson, Lancet, October 1980; see also Section 3.2 in Part A of this chapter) who strongly objected to the interpretation of these studies with respect to the predominant role of smoking. (That author again presented the data on cough, phlegm and breathlessness in the more than 11,000 British coal miners studied and showed clearly that "they do not support the claim that 'smoking is the main cause of symptoms'." He explained how the three-to-one ratio of the smoking effect (at age 65) to the maximum anticipated dust effect which had been used to justify the "main cause" argument with respect to smoking and FEV_1 loss was faulty, as first, many miners have experienced cumulative dust exposure substantially higher than in the study and that, secondly, the sample of miners in the study was atypical. He pointed out that even the original study drew attention to this factor and warned that it might have resulted in an underestimation of the effect of dust exposure. Further, he revealed that the follow-up study did show that the effect of dust exposure on the reduction of FEV_1 was possibly twice as severe as previously documented, while the reduction attributed to smoke was no more marked than previously revealed. In conclusion, he stressed that sweeping generalizastions about the relative importance of smoking and occupation on bronchitis are inappropriate.)

The failure of the Board-employed physician to reflect this article in his conclusions about this claim is consistent with the less than adequate "plant investigation" that accompanied it. The fact that the plant had a reputation for being extremely dusty was well-known as was the fairly large number of workers from this plant who had documented silica exposure on their x-rays. That this was not clearly reflected in the Board's investigation leads to the impression that the appraisal was inadequate.

Discussion:

As discussed in Section 2, the problem with silicosis in Ontario is not silicosis but chronic bronchitis. It was found that 11 of the 12 appealed rejections in the silicosis survey were actually cases of chronic obstructive lung disease. Some of the cases presented above are typical of the "silicosis rejections" of this sort. In Part A of this chapter it was noted that the number of occupations in which industrial bronchitis has been documented is on the increase. Nevertheless, the major reason given for rejecting the majority of WCB claims for chronic obstructive lung disease is that smoking alone could have been responsible for the respiratory impairment and therefore there was no need to hypothesize an industrial cause.

The number of claims of this nature is increasing as is the general awareness of respiratory hazards. Senior medical staff of the Board informed this writer that it planned a major study of Ontario industries to better assess the role of occupation in chronic obstructive lung disease. Perhaps this will help rectify the great injustice in the current pattern of adjudication of claims of this nature.

4. The Issues Summarized

1. As seemed to be the case for occupational cancer, there is reason to suspect that workplace factors play a larger role in non-malignant respiratory illness in the province than is currently reflected in Workers' Compensation Board statistics. Non-malignant respiratory disease accounts for significant morbidity and mortality in Ontario. As shown in studies discussed in Part A of this chapter, workers may not be incorrect in thinking that much of their respiratory disease is related to or aggravated by their workplace conditions. Nonetheless, only 311 Ontario workers received compensation for any component of their chest disability in 1981, less than half of which were for pneumoconioses, bronchitis, asthma or emphysema. Notwithstanding the fact that Ontario statistics for allowed respiratory disease claims are among the most impressive of any compensation jurisdiction, a fair number of workers with occupational respiratory disease seem to remain uncompensated.

2. The extent to which one agrees that there is significant under-compensation of occupational respiratory disease is largely determined by how one defines "occupational". Programs are in place to detect those with occupationally-specific diseases such as asbestosis or silicosis. However, much illness, work absence and suffering are due to non-specific respiratory diseases such as asthma, bronchitis and emphysema that are multifactorial in nature. Where workplace conditions caused or contributed to workers' non-specific respiratory diseases, the workers are generally not identified as having occupational respiratory disease.

3. Policy regarding these respiratory diseases has been difficult to formulate and claims are difficult to adjudicate, often because of confounding factors, particularly smoking. Unlike the majority of claims received by the Board, which take an average of less than two weeks to adjudicate, respiratory disease claims often take months to adjudicate. There was a mean of over 7 1/2 months before the claimant was notified of an initial

decision in the claims reviewed in the silicosis index. (As slow as this may seem, it compares favourably with other jurisdictions; in the U.S., industrial disease claims average over 1 year to be settled.)

4. Furthermore, whereas the rejection rate for claims overall is only a few per cent, the rejection rate for respiratory disease claims is over 50%. Respiratory disease claims that fall outside the schedule of diseases and specific guidelines, for example, for chronic obstructive lung disease (alpha-1-antitrypsin deficiency or sulphur dioxide exposure) seem only to be very rarely accepted. Although monitoring and identification of workers with occupational respiratory disease could be improved, current Board policy and procedure would be unlikely to produce a significantly greater number of successful claims.

5. The role of the "inherited predisposition" to develop respiratory disease seems to be dealt with by the Ontario WCB in what amounts to an inconsistent manner. For example, in comparing the adjudication of asthma claims with the policy regarding emphysema, it is apparent that if a genetic predisposition is found with respect to the former, only the discrete asthmatic episode in question is compensated and the individual is not eligible for wage-loss benefits if he or she is required to leave work due to this condition. In contrast, only those with documented specific genetic pre-dispositions to develop emphysema are eligible for benefits if the disease does indeed result. (It could, however, be argued that the two conditions are not comparable, as the residual damage in the asthmatic is debatable, whereas in the worker with emphysema the damage is, by definition, irreversible.) Nevertheless, the fact that genetics come into play at all can be seen as contrary to the spirit of "taking a worker as he or she is found".

6. Other sources of injustice to the injured worker, related to the previous point, lie in the eligibility criteria for total temporary (or wage loss benefits) both in the period of diagnostic work-up (for example, occupational asthma) and in the period after the diagnosis has been confirmed and relocation away from exposure is necessary or desirable. The very act of filing a compensation claim can be detrimental to the claimant in the long run, as once the employer is aware that an individual has health problems at work there has been nothing to protect the worker from a "medical layoff". Proposed amendments to the Workers' Compensation Act will, however, be addressing this matter.

7. Aside from the high rejection rate, the long delay, and the role of genetic susceptibility factors in case adjudication, even when the claim is allowed there is much dissatisfaction since the level of disability assessed as compensable is often considered by the claimant to be inadequate. Although the enhancement or aggravation effect is clearly taken into account in Ontario in the adjudication of injuries where pre-existing or underlying conditions exist, it is not clearly specified when, if ever, this principle is to be applied to respiratory disease claims. Far too little information is provided in the claim file as to the rationale for the percentage of partial disability decided upon. It certainly seems that often little consideration is given to the extent to which a compensable impairment aggravates or interacts with other medical conditions and much frustration is created for workers who are indeed totally unable to work, but for whom the occupational component of their disability has been assessed at 10 or 20 or 60 percent. The majority of claimants with

respiratory disease (73% of the allowed claims in the silicosis survey) never qualified for pension supplements at all and therefore were left only with the assessed impairment for the compensable condition. Presumably many of these claimants would benefit considerably from the recommendations of the 1981 White Paper or from any similar WCB reform proposals which instituted compensation for actual wage loss, although much depends upon how its principles are put into practice.

8. A separate issue from the assessment of disability in the presence of non-compensable medical conditions is the question of the multifactorial nature of a single respiratory condition. In this regard, we find that claims which are allowed in Ontario for chronic obstructive lung disease received benefits that ranged from 20% of the total assessed disability in a smoker to 40% in a non-smoker. It is certainly unclear what occupational factors make up the other 60% of the causation of this particular respiratory impairment. In any event, as these claimants would likely be eligible for wage loss benefits according to the White Paper recommendations arbitrary assessments of physical impairment would take on less importance.

9. The percentage of disability assessment has held even greater significance in the eligibility for fatal benefits. This issue will be addressed at length in the next chapter.

APPENDIX 1

Summary of the Evolution of Board Policy on Silicosis and Other Pneumoconioses

1915 - The W.C.A. came into effect.

1917 - "Miner's Phthisis" became a compensable disease under Schedule 3 of the Act.

(Interpretation clarified 1932 and deleted from the Act.)

1925 - "Grinder's Phthisis" became compensable (including quarrying, cutting, crushing, grinding or polishing stone or metal).

(Addressed in the Roach Report 1950.)

1926 - An amendment was passed naming "silicosis" as a compensable disease under the Act.

- "silicosis" referred only to the Mining Industry (amended in 1957)
- 5 years exposure was required (amended in 1944)
- the claims had to be filed within 1 year of cessation of exposure (amended in 1933, 1937, 1939, 1944 and repealed completely)
- a special "Silicosis Account" was established
- silicosis was defined to mean a fibrotic condition of lungs sufficient to produce a decreased capacity for work caused by the inhalation of silica dust (addressed in 1968).

1926 - A further amendment recognized pneumoconiosis (clarified in 1957).

1927 - A Board known as the Silicosis Referee Board was constituted to determine whether or not silicosis was present, to determine the amount of disability and to recommend a date for re-examination (role expanded 1929, 1952 and name changed 1966). (Its first members were from the Ontario Ministry of Health, aided by one or two consultants from the University of Toronto.)

1928 - Legislative provision was made under the Mining Act of Ontario requiring that every person employed in a dust exposure occupation in a mine must have a pre-employment examination and chest x-ray by a medical examiner appointed for the purpose.

- On January 1st, 1929, five Mining Chest Examination Stations were strategically located around the province. (Later another was added in Elliot Lake.) (These were under the Board's jurisdiction until 1975.)

1932 - Silicosis complicated by pulmonary TB with greater than one year, but less than five years exposure to silica dust in Ontario mines had been considered a case of "miners phthisis", where exposure was five years or more, it is considered complicated "silicosis" and thus paid from the Silicosis Account. Reference to miners phthisis being the same disease as TB complicated silicosis was deleted from the Act. (Active TB as a complication of silicosis results in automatic total disability regardless of previous rating - 1966.)

1933 - An amendment was passed limiting the time in which a claim could be filed to two years after employment had ceased.

1937 - The above was amended to three years.

1939 - The above was amended to five years.

1941 - The date of disablement was deemed not necessarily to be the date the worker ceased to be regularly employed in the industry of exposure, but rather the date the Board found he was disabled from earning full wages at the work at which he was employed. (Further clarified in 1975.)

1942 - The first case of asbestosis was accepted.

1944 - The period of exposure required for allowance of a claim was reduced from five to two years.

- The necessity of filing a claim within five years of leaving exposure became no longer required.

- A Residence clause remained (addressed by McGillivray in 1967).

1946 - Claimants for silicosis were put on a pension if they left the industry in which they were disabled. (Amended in 1974.)

1950 - The Honourable Justice W.D. Roach recommended eliminating separate reference to stone workers' and grinders' phthisis as unnecessary as it was included by the term "silicosis," and recommended broadening the wording with respect to the industrial processes which may be involved. Further issues were considered but resulted in no concrete recommendations.

1950 - A medical statistics unit was established at the WCB at the suggestion of the Ministry of Health to analyze data on the incidence, prevalence and progression of silicosis in the mining industry. (This was later computerized and became the source of data for the Ham Commission and for the Ontario Miners Mortality Study.)

1951 - The Board ordered that where a lump sum award for silicosis in an "ante-primary stage" of $500 or in the "primary stage" of $1,000 has been made, it was to be disregarded when the claim was reopened and an award made for silicosis by way of a monthly pension.

1952 - Examination of employees exposed to silica in industries other than mining become compulsory in Ontario. The Silicosis Referee Board expanded its role to include exposed workers in foundries, etc.

1955 - The Board ordered that the Silicosis Referee Board should state the percentage rating over 25% and where the disability is under 25% the report is to state "silicosis established" disability is not presently compensable. The order stated that the percentage ratings were to be based on clinical findings, but where special consideration is considered warranted, the circumstances are to be outlined. (Amended 1968.)

1957 - An amendment eliminated the restriction of the use of the term "silicosis" to the mining industry and pneumoconiosis was clarified to refer to non-silica dust disease of the chest.

1958 - The Board ordered that rehabilitation of silicotics whose exposure was subsequent to the year 1928 was to be considered as soon as possible after the claimant was determined to be a silicotic in Category 5. (Addressed in 1966 and 1968.)

1966 - Letter from Dr. Sutherland, Director of the Industrial Hygiene Branch to the Ontario Mining Association suggesting that funding be allowed for rehabilitation and retraining of early silicotics (prior to disability and entitlement to compensation).

1967 - Honourable Justice McGillivray recommended that legislation be enacted to authorize the Board to make agreements with other provincial WCBs in Canada with respect to claims where there has been exposure to silica dust in more than one province. Other issues considered, but no specific recommendations articulated.

1968 - Board approved the allowance of a claim with less than a 25% rating to entitle the claimant to benefits and specifically to rehabilitation assistance.

1970 - The name of the Silicosis Referee Board was changed to the Advisory Committee on Occupational Diseases to better reflect its function. The 7 members were drawn from the Ministry of Labour, Ministry of Health, University of Toronto and McMaster University.

1974 - Pension awards became payable if pneumoconiosis was diagnosed whether or not the employee remained in the industry.

1975 - As a result of the recommendations in the Report of the Task Force WCB (April 1973), the operations of the Chest Examination Stations were transferred from the Board to the Ministry of Health.

1975 - It was determined that the date of disability would be the date at which the x-ray first showed silicosis (i.e. the initial examination by the A/C confirmed the diagnosis or the date of subsequent examination by the Advisory Committee at which time the diagnosis was subsequently established or earlier as recommended by the Advisory Committee).

1975 - The Board directed the "save harmless" principle for mines hiring at Elliott Lake mines with "code 4" x-rays should not be extended generally to apply to miners who develop a "4" rating in mines other than Elliot Lake. If a miner appeals this decision, it should be dealt with on the individual merits of the situation.

1975 - A rehabilitation program came into being. However, its application was only to those with present impairment, except in situations covered by the Board's Special Vocational Rehabilitation Program. (This covered Elliot Lake mines with "dust effects.")

1976 - The rehabilitation program was extended to asbestos workers with "dust effects."

1977 - Cost charging for silicosis claims was reviewed; the Board reiterated its position with respect to charging the last employer and the use of the Silicosis Fund rather than the SIEF.

APPENDIX 2

Pulmonary Function Tests and Disability Assessments

It was noted in Part A of this chapter that pulmonary function tests are the cornerstone of the assessment of respiratory impairment. Consequently, an attempt was made in this study to correlate pulmonary function test results with disability awards by the WCB. Unfortunately, inadequate data were included in the claim files to be able to perform this evaluation. A great many claim files merely had summary statements or conclusions of the Advisory Committee on Occupational Chest Diseases (ACOCD) without providing the numerical test results on which they were based. Where available, some pulmonary function test results have been provided in the text. The results for claimant 40 (Case 1, Section 2.3) were provided in the file and are therefore tabulated here. The meaning of these parameters was discussed at length in Part A of this chapter. In taking into account the chest x-ray findings as well as these pulmonary function results, the ACOCD concluded that the claimant had moderate obstruction and slight restrictive pulmonary disease; he was awarded a 10% permanent disability award.

Pulmonary Function Test Results for Claimant 40

	1978				1981	
Test	Actual: Pre-Exercise	% Predicted	Actual Post-ex.	% Predicted	Actual (pre-dilator)*	% Predicted
FVC	2.76	66	2.38	57	2.99	72
FEV_1	1.74	55	1.66	52	1.88	60
FEV_1/FVC	63.0	-	70.0	-	63.0	-
FEF_{25-75}	0.85	27	1.04	33	.75	24
FEF_{75-85}	0.18	24	0.18	24	N/A	N/A
$V_{max\ 50\%}$	0.24	11	1.47	35	0.22	30
VC	3.29	79			3.47	83
TLC	5.53	89			5.80	94
IC	2.78	77			2.82	78
FRC	2.75	106			2.98	115
RV	2.23	130			2.33	135
$RV/_{TLC}$	40	-			40	-
TL_{CO}	29.6	127			23.1	98
IC_{CO}	4.0	112			4.1	116

* Worse post-dilatation

APPENDIX 3

WCB POLICY AND REVISED POLICY CONCERNING ALLERGIC RESPIRATORY DISEASE

A. Conclusion of Committee on Allergic Disease in Industry

(March 30, 1962)

Conclusions are as follows:

1. Antibody formation is a natural defence mechanism of the body against invading allergens. When this defence is over-activated allergic illness results. The commonest manifestations of such illness involve skin and the respiratory system. Almost all individuals can be sensitized if the allergen is sufficiently potent and the exposure is adequate.

2. The cause of the allergic response is not well understood.

3. It has not been established that permanent biochemical changes occur in the cells following an allergic illness. However, certain structural changes in organs are recognized, e.g. emphysema following asthma, lichenification of the skin following contact dermatitis.

4. Heredity and age are factors which predispose to allergic disease. It is not practical to exclude from industry, individuals in whom these factors are active. Therefore, neither age nor heredity per se warrant limitation of entitlement under the Workmen's Compensation Act, Ontario.

5. (a) Although an episode of allergic disease precipitated by one allergen may make an individual more susceptible to a similar illness provoked by unrelated allergens, this relation is very difficult to establish or disprove.

   (b) Such episodes are to be considered causally related only if the earlier condition is still active at the time of onset of the subsequent illness. Thus, if the initial cause is occupational and later illness results from non-occupational factors, entitlement under the Workmen's Compensation Act, Ontario, is limited to the point of recovery from the occupational episode.

   (c) The converse is also applicable, namely in an individual with known non-occupational sensitivities, these are to be considered causally related only if acting at the time of onset of the occupational episode.

   (d) Where known prior non-occupational allergic disease is reactivated by a related allergen in the employment, entitlement is terminated with recovery from the acute episode. Where recovery does not occur from this acute episode, a permanent disability award can be considered.

6. Therefore, it is recognized that a workman may suffer from an allergic response, either respiratory or dermatological, to certain elements to which he is exposed in the employment. Within the above qualifications he has under the Workmen's Compensation Act, Ontario, entitlement for treatment.

7. If a workman does not show improvement within six months after removal from exposure to the offending allergen, and is still suffering from acute episodes, the

claim should be referred for assessment as to continuing entitlement to this Board's specialist in dermatitis or respiratory disease. This lack of response may be due to significant structural change or to perpetuation by non-occupational allergens.

8. When it is established that maximum recovery has been achieved, the claim should be brought to settlement.

9. If the compensable episode is stabilized and the workman is receiving treatment on the preventive level, there would usually be no entitlement for medical aid once the permanent disability award is made.

10. If the compensable episode is stabilized and the workman still has significant disability, he should be considered for a permanent disability award:

    (a) Date of permanent disability rating should be at least six months after removal from exposure, or from date of last exacerbation.

    (b) To facilitate consistent evaluation, this Committee recommends that all permanent disability ratings be carried out by the Senior Permanent Disability Medical Officer, the Workmen's Compensation Board's specialist in the respective field, and a Permanent Disability Rating Officer.

11. In older persons who have recovered from the acute illness, but who are forced to leave certain employment to avoid further exposure, who cannot do other work and must suffer wage loss, an Industrial Rating may be given without Medical Aid for further treatment.

B. Revisions to Policy on Allergic Respiratory Disease

(1963 and 1965)

Order 14

1. Every claim for allergic respiratory illness is to be referred for adjudication to the Industrial Disease Medical Officer in consultation with the Head of the Chest Services Department.

    (a) Local investigation as to exposure, examination at the Board's Toronto office, or referral to a consultant may be ordered.

2. In prolonged claims where a workman does not show improvement within three months after removal from exposure to the offending allergen and is still disabled from work, the claim is to be referred to the Industrial Diseases Medical Officer in consultation with the Head of the Chest Services Department for assessment of continuing entitlement and to determine if any portion of the workman's continuing disability is attributable to a condition or conditions not included in his entitlement under the Act.

3. Where it is established that maximum recovery has been achieved, the claim is to be brought to settlement.

(a) A permanent award is to be considered where the compensable episode is stabilized and at least six months has elapsed after removal from exposure or from the date of the last exacerbation.

(b) To facilitate consistent evaluation, every clinical permanent disability rating is to be recommended by the Senior Permanent Disability Medical Officer in consultation with the Head of the Chest Services Department. (May 14, 1965)

4. At the time of rating, the nature and extent of continuing medical aid is to be defined by the Head of the Chest Services Department and the principle of the settlement explained to the workman.

(May 9, 1963)

APPENDIX 4

Guidelines for the Adjudication of Claims for Pulmonary Emphysema due to Alpha-1-Antitrypsin Deficiency
June 1979

1.0 Emphysema related to alpha-1-antitrypsin deficiency, when identified by tests in those occupations as outlined in this document (Section 8), will be accepted as disablement arising out of the employment under Section 1(1)(a)(iii) of the Act.

2.0 Alpha-1-antitrypsin deficiency is an acceleration or activation factor in the development of pulmonary disability resulting from emphysema. Claims relating to this disability will be favourably considered when all the following circumstances apply:

2.1 acceptance be dependent on objective medical evidence of emphysema with impairment;

2.2 the duration of occupational exposure shall be 15 years or more;

2.3 the pulmonary disability appears between the age of 30 and 60 years;

2.4 proof of presence of ZZ or MZ phenotype by testing.

3.0 Temporary Total Disability

3.1 Temporary total disability benefits under the provisions of Section 39 of the Act are not payable for periods of acute pulmonary disease in the absence of an award under Section 42(1) of the Act.

3.2 Temporary total disability may be paid under the following circumstances:

3.2.1 where surgical interference is warranted in the presence of a confirmed diagnosis and disability award;

3.2.2 during the definitive medical investigation preceding the confirmation of diagnosis;

3.2.3 during acute lower respiratory illness where indicated, subject to appropriate medical evaluation.

4.0 Permanent Disability Award

4.1 Smoking history and phenotype are to be taken into consideration when establishing percentage relationship to permanent disability. The percentage of the permanent disability accepted by the Board shall be:

	Phenotype	**Relationship - Percentage of Disability Accepted**
Smoker	ZZ	25%
	MZ	50%
Non-smoker	ZZ	50%
	MZ	100%

5.0 Claims involving **EX-SMOKERS** are to be evaluated having regard to:

5.1 the length of cessation interval;

5.2 the chronological relationship of such cessation interval to the onset of objective symptoms and/or manifest pulmonary impairment.

Definition of Smoking

1. ***Non-smoker***--a never smoker. A person who has never smoked even occasionally.

2. ***Ex-smoker***--a person who has smoked during his lifetime but who has not smoked for the past ten or more years.

3. ***Smoker***--a person who currently smokes, or who has ceased smoking for less than the previous ten years.

6.0 Proportionate Disability

6.1 Proportionate disability shall apply for permanent disability awards with no cost transfer to the Second Injury and Enhancement Fund.

6.2 Full benefits may be paid under Section 39 and 36 when they apply with a cost transfer to the SIEF based on the proportionate percentage established for the permanent disability award.

(Example: If permanent disability is for 25% then 75% of temporary total disability compensation and medical aid for the period will be transferred; similarly, 75% of benefits granted under Section 36 will be transferred.)

Claims which do not meet the above criteria shall be individually judged on their own merits having regard for the evidence submitted. The benefits of reasonable doubt applied.

7.0 The following industrial processes or occupational exposures may be considered hazardous to those with the proven genetic deficiency:

7.1 underground mining;

7.2 firefighting;

7.3 coke oven (topside or side oven);

7.4 welding;

7.5 metal refining (to include zinc, copper cadmium, nickel and precious metals):

7.5.1 roasting;

7.5.2 smelting;

7.5.3 sintering;

7.5.4 converting;

7.6 foundry:

    7.6.1 moulding;

    7.6.2 knockout;

    7.6.3 fettling;

    7.6.4 sandblasting;

    7.6.5. grinding;

    7.6.6 core room;

7.7 pulp and paper;

7.8 surface dust operations (to include milling, crushing, grinding, cutting and drilling):

    7.8.1 silica;

    7.8.2 asbestos;

    7.8.3 talc;

    7.8.4 nepheline syenite;

    7.8.5 raw cotton;

7.9 prolonged exposures involving, for example, chlorine, acid mist, ammonia, oxides of nitrogen, metallic fume, sulphur dioxide and ozone;

7.10 any operation which may be developed in the future involving regular exposure to irritant dust, gas vapour or fume and not covered in previous paragraphs.

APPENDIX 5

Guidelines for Adjudication of Claims for Chronic Obstructive Lung Disease (COLD) in Smelter Workers and Related to Sulphur Dioxide (SO_2) and Particulates Exposure

1.0 COLD in smelter workers shall be accepted as an industrial disease under Section 118 and Section 1(1)(L) of the Act as peculiar to and characteristic of exposure to SO_2 and particulates.

2.0 Based on medical studies, claims for COLD with impairment shall be favourably considered when all the following circumstances apply regarding exposure, intensity, latency (inception period), and cessation factors:

2.1 Nature of Exposure

Any smelting process involving the production of sulphur dioxide and particulates as a significant by-product of the process may be considered a causative factor. This would include the roasting, the smelting and converting of metallic ores and the secondary production of sulphuric acid.

2.2 Intensity of Exposure

Sulphur dioxide exposure is established when levels of more than five parts per million (5 p.p.m.) have been recorded repeatedly over the exposure period as specified in 2.3

2.3 Duration of Exposure

A minimum of 20 years established exposure as defined under 2.1 and 2.2

2.4 Inception Period (Latency)

A minimum of 20 years from date of first exposure.

2.5 Cessation Interval

Where the cessation of risk is less than five years to the development of clinically disabling COLD, the presumption of SO_2 effect shall apply.

3.0 Acceptance of claims shall be dependent on objective medical evidence of COLD and impaired pulmonary function as identified by objective tests.

4.0 A diagnosis of primary bronchiectasis, chronic upper respiratory disease or primary asthma without the presence of COLD shall not be considered an acceptable basis to claim under these guidelines.

5.0 Smoking history shall be taken into consideration when establishing **percentage relationship to permanent disability.** The percentage of permanent disability accepted by the Board shall be:

5.1 **Smoker**—20 percent of the assessed P.D.;

5.2 **Ex-Smoker**—30 percent of the assessed P.D.;

5.3 **Non-Smoker**—40 percent of the assessed P.D.

Definition of Smoking

1. Smoker

A person who currently smokes, or who has ceased smoking for less than the previous 10 years.

2. Ex-Smoker

A person who has smoked during his lifetime, but who has not smoked for the past 10 or more years.

3. Non-Smoker

A person who has never smoked, even occasionally.

6.0 Temporary Total Disability

6.1 Temporary total disability under the provisions of Section 39 of the Act is not payable for periods of acute pulmonary disease in the absence of an award under Section 42(1) of the Act, except if required during the definitive medical investigation in order to confirm the diagnosis, when so ordered by the Board.

6.2 Temporary total disability may be paid in established claims for Permanent Disability (Section 42(1)). Such payment shall be for periods of disability due to acute lower respiratory illness subject to appropriate medical evaluation.

7.0 Permanent Disability

7.1 Proportionate Disability shall apply for Permanent Disability Awards with no cost transfer to the Second Injury and Enhancement Fund (SIEF).

7.2 Full benefits may be paid under Sections 39 and 36 when they apply with a cost transfer to SIEF based on the proportionate percentage established for the Permanent Disability Award. (EXAMPLE: If Permanent Disability is for 20% then 80% of Temporary Total Disability Compensation and Medical Aid for the period will be transferred. Similarly, 80% of benefits granted under Section 36 will be transferred.)

Claims which do not meet the above criteria shall be individually judged on their own merits having regard for the evidence submitted. The benefit of reasonable doubt applied.

CHAPTER IV

WORK-RELATED DEATH: THE ADJUDICATION OF CLAIMS FOR SURVIVOR'S BENEFITS AT THE ONTARIO WORKERS' COMPENSATION BOARD

1. An Analysis Of Death Benefit Statistics

1.1 Overview

Death of a family member, regardless of cause or age of occurrence, is surrounded by grief and often anger. It usually leaves the survivors with a host of new problems to sort through. All too often financial insecurity looms in the background and amidst the sorrow a quest for retribution may emerge. Accordingly the adjudication of claims for survivor's benefits tends to be a very sensitive area, and one that receives considerable attention at the Ontario Workers' Compensation Board. The previous three chapters outlined the difficulties in determining work-relatedness of disease. As would be expected, the work-relatedness of death is a source of extreme contention. A careful appraisal of the handling of claims for survivor's benefits by the WCB is therefore warranted, with a view to understanding where the controversy exists, why it exists and what could be done to resolve it.

As shown in Table IV-1, claims were filed for 531 workers who died in 1980. Of these, 343 (64.6%) were "new" claims, initiated after the death of the worker, with no temporary or permanent disability benefits having been previously awarded; and 188 (35.4%) were "old" claims that is, the deceased had been receiving compensation at the time of death. Two-thirds of the "new" claims were fatalities due to industrial accidents. Thus the first point that emerges is that the commonly thought-of "industrial fatality", death in the course of an industrial accident, accounts for less than 60% of survivor's benefit claims filed.

The second point that emerges from the data in Table IV-1 is that while 81.5% of "new" fatal accident claims were allowed, only 15.3% of "new" claims for fatal diseases were allowed. Most of these claims (44.5%) were for cancer (as discussed in Chapter II) with cardiac claims accounting for another 36.1%. (These claims will be discussed in more detail in Section 3 below.)

More importantly, in the "old" claims that were filed, the cause of death was deemed to be related to the compensable condition in 43.2% of the trauma-disabled and in 56.7% of the disease cases. Thus although the rejection rate and length of adjudication time in non-fatal compensation claims due to accidents is very different from those in disease claims (as was discussed in the previous chapters), it seems that the outcomes regarding the work-relatedness of death in these two sets of claims are fairly similar. Section 43(7) of the Act creates an essentially irrebuttable presumption in favour of compensating the survivor where the worker is permanently and totally disabled by a compensable illness or injury prior to death. In other words, the survivors are automatically entitled to compensation where the deceased was receiving a 100% P.D. award at the time of death regardless of the cause of death. In contrast, where a worker is considered less than 100% permanently disabled by a

TABLE IV-1

1980 Survivor's Benefit Claims by Claim Status and Category[a]

	Allowed	Rejected	Other[c]	Total
Old Claims[b]				
Accident	42	46[d]	-	88
Disease	55	43[d]	2	100
Total	97	89	2	188[d]
New Claims[b]				
Accident	189	21	22	232
Disease	17	92	2	111
Total	206	113	24	343
Grand Total	**303[e]**	**202**	**26**	**531**

Notes

a These data were compiled from the fatal claims card index maintained by the Policy Planning Secretariat (formerly P.P. & S.S. Division).

b "Old" claims refer to cases in which the worker had been receiving compensation prior to death; "new" claims refer to claims filed after the death of the worker.

c Claims in which "no further action" was required (e.g. no dependents, or claim was withdrawn).

d Includes 26 claims in which the cause of death was deemed not work-related but for which survivor's benefits were awarded according to the Act as the deceased had been receiving a 100% P.D. award. (These 26 claims are usually excluded from official statistics as they are neither work-related deaths nor rejected from the point of view of the claimant.)

e The official statistics of allowed claims for survivor's benefits in 1980 were published in March 31, 1981 and listed this total as 272. Between that time and the time of this survey (Fall, 1982) more claims had been allowed on appeal and pending claims have since been settled.

compensable disease (or injury) a judgment must be made by the medical staff regarding the cause of death. Understandably this matter has proven highly controversial. A more detailed analysis of these ("old") claims is therefore undertaken below.

1.2 *Compensation Award at Time of Death as a Predictor of Outcome of Claims for Survivor's Benefits*

As shown in Table IV-2, in 60 of the 188 "old" claims, the deceased had been receiving a total temporary disability (T.T.) award at the time of death and had never been assessed for a permanent disability (P.D.) award. The length of time on T.T. benefits ranged from a few days to almost 2 years. In the majority of these cases (71.6%), the cause of death was deemed to be related to the compensable condition and consequently survivor's benefits ensued. (Refer to Table IV-6 for the compensable conditions and causes of death.) While it seems that these claims are generally less contentious, several well-publicized exceptions will be discussed in Sections 2 and 3 below.

Survivor's benefit claims for workers receiving 100% P.D. awards also accounted for 60 claims, of which only 34 were deemed by the Board to be truly work-related deaths. (As illustrated in Table IV-6, in 9 of these the cause of death was unknown and in 4 others, a review of the claim showed that the work-relatedness of the death was never determined. Inclusion of these 13 claims artificially increases the percentage of deaths deemed related to the compensable condition, but does not significantly alter the results.)

While 43 of the 76 deaths (56.5%) in those survivors' claims where the worker had received a P.D. award over 60% were attributed to the compensable condition, only 11 of the 51 claims for survivor benefits filed for those with P.D. awards under 60% (21.6%) were allowed. Although it might be expected that the higher the P.D. award the more likely the compensable condition would be deemed to account for the death, this trend was not as marked as may have been expected. In fact, no clear correlation existed between the amount of the award and the likelihood that the claim would be allowed. The issues raised in Chapter III about the inaccuracy of disability assessments are of interest in this regard. It would certainly seem that the amount of the P.D. award is not an adequate predictor of the compensability of death and thus would cast doubt on a system which allowed survivor benefit claims on the basis of previous assessments of extent of compensable impairment.

Table IV-3 summarizes the data from a listing of survivors' claims from January to July 1981 compiled by a senior physician at the WCB for the Weiler Inquiry. It showed even more clearly that very few claims for survivor's benefits are allowed for workers who received P.D. awards of less than 100%. In fact, only 2 claims in this category were allowed, while 28 were rejected. The 2 allowed claims were on behalf of a worker who had been receiving a 5% P.D. award for an inguinal hernia and who died during the course of surgery, and a worker who had been receiving a 70% P.D. award for small airways disease and died of cardiac arrest. This claim was presented in Section 3.3 of Chapter III-Part B. It was allowed on the assumption that the claimant would have been 100% disabled at the time of death.) Among the claims allowed for those who had been receiving 100% P.D. awards for deaths deemed unrelated to the

TABLE IV-2

1980 Survivor's Benefit Claims (Traumatic & Disease - Year of Death 1980) By Permanent Disability Award at Time of Death and Claim Status[a]

	No. PD Award	**PD Award Percent**						
Survivor's Benefit Claim Status	**Some TD**	**20%**	**20-39%**	**40-59%**	**60-79%**	**80-99%**	**100%**	**Total**
(1980 Year of Death)								
Number of survivors' claims filed	60	13	19	19	16	1	60	**188**
Number of survivors' claims allowed	43	4	4	3	8	1	34	**97**
Number of survivors'[b] claims rejected	17	9	15	15	7	-	26	**89** [b]
Other status (withdrawn, N.F.A., etc.)		-	-	-	1	1	-	**2**

Notes

[a] Excludes "new" claims; see note b in Table IV-1.

[b] For 100% P.D. awards the number allowed represents the number deemed work-related or in whom the cause of death was unknown. In 26 fatal claims for workers who had been receiving 100% P.D. awards, the cause of death was deemed unrelated. These appear as "rejected" claims for purposes of this analysis.

TABLE IV-3

Claims for Survivor's Benefits January 1 - June 30, 1981[a]

	Death Deemed Related to Occupational Factors or Compensable Disease	Death Unrelated to Occupational Factors or Compensable Disease	Other
Total temporary disability at time of death or newly-established claim for survivor's benefits.	19	3 [a]	3
Previous claim rejected new claim established at time of death.	-	13	-
Claimant receiving a P.D. award of less than 100% at time of death.	2 [b]	28	
Claimant receiving 100% P.D. award at time of death.	18	20 [c]	

Notes

a This table was compiled from a list provided by the W.C.B. industrial medical consultants, in which an attempt had been made to exclude "new" claims that were deemed unrelated to employment. Three such claims were included in the list in error.) The total number of "new" claims filed in this period is therefore not provided as it would be misleading. The total number of "old" claims in this 6 month period was 88, corresponding to what would be expected from the 1980 totals.

b Many other claims would have been in this group but were converted from P.P.D. awards to 100% after death in order to provide entitlement to survivor's benefits. This probably explains the difference between these data and those in Table IV-2. More importantly, it underlines the importance of the presumption afforded by Section 42 (7) of the Act.

c Benefits were provided to survivors in these cases as dictated by Section 42 (7) although the death was considered not work-related.

compensable condition, a sampling of the claims revealed that claims are often converted to 100% P.D. after the worker dies in order to provide entitlement.

Even if reform measures were introduced to base workers' compensation benefits upon actual wage losses (instead of physical impairment) a survivor's entitlement to WCB benefits would depend upon his or her ability to prove that the worker's death was occupationally related. Thus, the determination of the work-relatedness of the death, rather than the P.D. award, would be of paramount importance in any case. The fact that death was deemed not work-related in almost half of the claimants receiving 100% P.D. awards is potentially problematic in this regard. The issue will be examined in more depth in Section 1.5 with respect to cause of death and compensable condition.

1.3 *Outcome of Claims for Survivor's Benefits by Compensable Condition*

As shown again in Table IV-4, the greatest number of claims for survivor's benefits were filed by the survivors of workers who had been receiving compensation (i.e. "old" claims) for industrial accidents (46.8%). Workers disabled by occupational respiratory disease accounted for 29.7% of the "old" fatal claims, and victims of occupational cancer for another 17%.

As would be expected, the vast majority (96.8%) of those receiving compensation for cancer died of this disease. In contrast, in 37.5% of the compensated cardiac claims and in 33.9% of the occupational respiratory disease cases, (for which survivor's benefit claims were filed) the cause of death was not considered related. Death was considered work-related in 47.7% of the survivors' claims filed for those who had been disabled by traumatic injuries. Excluding the "T.T.-only" claims, in 6 of 7 cancer claims, 28 of 60 trauma claims, 2 of 3 cardiac claims and 18 of 55 respiratory disease claims, death was considered by the WCB to be related to the compensable condition. Thus, according to the WCB, most people do not die of their compensable condition. The largest group of survivor's benefit claims for workers who had suffered from occupational diseases were on behalf of those with respiratory disease. These claims will be considered at length in Section 2.

1.4 *Outcome of Claims for Survivor's Benefits by Cause of Death*

As shown in Table IV-5, the cause of death that accounted for the largest number of benefit claims filed by survivors was cancer (26.6%). This was followed by cardiac disease (25%) and respiratory disease (20.7%). Other illnesses accounted for 14.4% of the survivor's benefit claims filed. The cause of death was unknown in 8.5% and traumatic deaths accounted for only 4.7%. Not surprisingly, the major causes of death in the population at large also accounted for the death of individuals on compensation.

As claims from those on T.T. are more akin to new fatal claims and are not associated with the same issues in determining compensability of the death, an assessment of the data excluding this category may be more appropriate in evaluating the problem areas in adjudication. On so doing it is clear that cardiac and respiratory deaths account for the largest number of deaths in individuals receiving P.D. awards. (Due to the poor prognosis associated with cancer, most workers who contract occupational cancer are never assessed for P.D. awards, but receive

TABLE IV-4

1980 Claims for Survivor's Benefits (Traumatic & Disease - Year of Death 1980) By Permanent Disability Award, Compensable Condition and Claim Status[a]

Compensable Condition & Status of Claim	No. P.D. Award	Percentage of P.D. Award						
For Survivor	Some T.D.	20%	20-39%	40-59%	60-79%	80-99%	100%[b]	Total
Traumatic								
Allowed	14	2	-	1	3	1	21	42
Rejected	14	7	4	4	3	-	14	46
Other	-	-	-	-	-	-	-	-
Cardiac								
Allowed	3	1	-	-	-	-	1	5
Rejected	2	-	-	-	-	-	1	3
Other	-	-	-	-	-	-	-	-
Respiratory								
Allowed	1	-	2	1	5	-	10	19
Rejected	-	1	11	10	4	-	1	37
Other	-	-	-	1	1	-	-	2
Malignancy								
Allowed	25	1	2	1	-	-	2	31
Rejected	-	-	-	1	-	-	-	1
Other	-	-	-	-	-	-	-	-
Other Industrial Illnesses								
Allowed	-	-	-	-	-	-	-	-
Rejected	1	1	-	-	-	-	-	2
Other	-	-	-	-	-	-	-	-
Total	60	13	19	19	16	1	60	188
Allowed	43	4	4	3	8	1	34	97
Rejected	17	9	15	15	7	-	26	89
Other	-	-	-	1	1	-	-	2

Notes

a,b See notes for Table IV-2.

TABLE IV-5

1980 Survivor's Benefit Claims (Traumatic and Disease - Year of Death 1980) By Permanent Disability Award, Cause of Death and Claim Status[a]

Cause of Death &	No.P.D. Award	Percentage of P.D. Award						
Survivor's Claim Status	Some T.D.	20%	20-39%	40-59%	60-79%	80-99%	100%[b]	Total
Traumatic								
Allowed	7	-	-	-	1	-	-	8
Rejected	-	-	-	-	-	-	1	1
Other	-	-	-	-	-	-	-	-
Cardiac								
Allowed	4	1	1	1	3	-	6	16
Rejected	7	3	4	5	1	-	10	30
Other	-	-	-	1	-	-	-	1
Respiratory								
Allowed	5	1	2	-	4	1	8	21
Rejected	2	2	5	1	2	-	6	18
Other	-	-	-	-	-	-	-	-
Malignancy								
Allowed	24	1	1	2	-	-	4	32
Rejected	2	2	4	4	1	-	5	18
Other	-	-	-	-	-	-	-	-
Other Illnesses								
Allowed	3	1	-	-	-	-	7	11
Rejected	5	2	2	4	3	-	-	16
Other	-	-	-	-	-	-	-	-
Unknown								
Allowed	-	-	-	-	-	-	9	9
Rejected	1	-	-	1	-	-	4	6
Other	-	-	-	-	1	-	-	1
Total								
Allowed	60	13	19	19	16	1	60	188
Rejected	43	4	4	3	8	1	34	97
Other	-	-	1	1	-	-	2	

Notes

a,b See notes for Table IV-2.

temporary disability (T.D.) benefits until their deaths; often, in fact, they only come to the attention of the WCB after death.) (It should be noted that the causes of death were derived by coders in the Policy Planning Secretariat - formerly Program Planning and Statistical Services Division, largely from death certificates. These in turn may be the source of considerable inaccuracy as discussed elsewhere in this report. Several of the cases presented later in this chapter amend some of the data presented in the Tables. None of these changes, however, alter the conclusions that may be drawn from the data.)

1.5 *Cause of Death by Compensable Condition*

It is informative to reflect upon the cause of death in workers who had been disabled by various occupational diseases or injuries. (Various epidemiological studies illustrating this point were referred to in Chapter III-Part A.) The most noticeable feature of Table IV-6 is that no worker who had been disabled by an industrial disease died as a result of trauma. This is what would be expected. It is also readily apparent from Table IV-6 that most (87%) workers with occupational cancer die of their disease.

From the top row in Table IV-6 it may, at first glance, seem surprising that compensation was allowed in 16 benefit claims by the survivors of workers who had been disabled by industrial accidents in which the cause of death was a disease of cardiac or respiratory origin; it may even seem more surprising that 2 claims were allowed for individuals who died of cancer. However a closer look shows that 5 of the 16 cardio-respiratory causes occurred in the initial period following the accident presumably precipitated by the compensable event, and 6 others were for individuals receiving 100% P.D. awards. Thus survivor's benefits were allowed for only 5 individuals who died of cardio-respiratory diseases and who had been receiving P.D. awards for disability resulting from industrial accidents. These 5 included: 1) Claim # 71 in which the claimant had sprained his ankle in 1976 and developed deep vein thrombosis which gave rise to thrombophlebitis and a pulmonary embolus. He had been receiving a 10% P.D. award for the recurrent thrombophlebitis which ensued. When the claimant died from a secondary pulmonary embolus in 1980, the claim was initially rejected as he had not had an active episode of thrombophlebitis since 1977. On appeal survivor's benefits were allowed in light of conflicting medical evidence. 2) Claim # 72. This worker had been receiving a 5 % award for a hernia and died of cardiomyopathy. Despite the fact that this underlying disease had been diagnosed at least 3 years earlier, the fact that the fatal cardiac arrhythmia occurred in the post operative period persuaded the WCB to allow the survivor's benefit claim. 3) Claim # 73. This claimant who died of a myocardial infarction had been receiving a 65% P.D. award resulting from injury to his legs. He was actually receiving T.T. benefits for bilateral below knee amputations at the time of his death. As it was concluded that he would have been considered 100% disabled had he been examined prior to his death the claim for survivor's benefits was allowed. 4) Claim # 74. This worker had been receiving a 60% P.D. award, having sustained multiple injuries when he was hit by a 500 pound weight. An abdominal aneurysm was diagnosed after the incident and was considered compensable "on an aggravation basis." However, when the claimant died due to a rupture of this aneurysm the Board rejected the claim for survivor's benefits stating that this was consistent with the natural history of aneurysms. On appeal

TABLE IV-6

1980 Death Benefit Claims (Traumatic and Disease - Year of Death 1980 By Cause of Death, Compensable Condition and Claim Status[a,b]

Compensable Condition and Fatal Claim Status	Cause of Death TRAUMA TT	TRAUMA PDF 100%	TRAUMA PD 100%	TRAUMA Total	CARDIAC TT	CARDIAC PDF 100%	CARDIAC PD 100%	CARDIAC Total	RESPIRATORY TT	RESPIRATORY PDF 100%	RESPIRATORY PD 100%	RESPIRATORY Total	MALIGNANCY TT	MALIGNANCY PDF 100%	MALIGNANCY PD 100%	MALIGNANCY Total	OTHER ILLNESSES TT	OTHER ILLNESSES PDF 100%	OTHER ILLNESSES PD 100%	OTHER ILLNESSES Total	UNKNOWN TT	UNKNOWN PDF 100%	UNKNOWN PD 100%	UNKNOWN Total	
TRAUMA																									
allowed	7	1	-	8	1	3	4	8	4	2	2	8	-	-	2	2	2	1	6	9	-	-	7	7	42
rejected	-	-	1	1	5	6	7	18	1	4	3	8	2	3	2	7	5	4	1	10	1	1	-	2	46
other	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
CARDIAC																									
allowed	-	-	-	-	3	1	1	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5
rejected	-	-	-	-	1	-	-	1	1	-	1	2	-	-	-	-	-	-	-	-	-	-	-	-	3
other	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
RESPIRATORY																									
allowed	-	-	-	-	-	1	1	2	-	6	6	12	1	1	-	2	-	-	1	1	-	-	2	2	19
rejected	-	-	-	-	-	7	3	10	-	6	2	8	-	7	3	10	-	6	3	9	-	-	-	-	37
other	-	-	-	-	-	1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	2
MALIGNANCY																									
allowed	-	-	-	-	-	1	-	1	1	-	-	1	23	3	2	28	1	-	-	1	-	-	-	-	31
rejected	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1[c]	-	1	-	-	-	-	-	-	1
other	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
OTHER OCCUPATIONAL DISEASE																									
allowed	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
rejected	-	-	-	-	1	-	-	1	-	-	-	-	-	-	-	-	1	-	-	1	-	-	-	-	2
other	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
TOTALS																									
allowed	7	1	-	8	4	6	6	16	5	8	8	21	24	4	4	32	3	1	7	11	-	-	9	9	97
rejected	-	-	1	1	7	13	10	30	2	10	6	18	2	11	5	18	6	10	4	20	1	1	-	2	89
other	-	-	-	-	-	1	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	2
TOTAL	7	1	1	9	11	20	16	47	7	18	14	39	26	15	9	50	9	11	11	31	1	2	9	12	188

Notes

a,b See notes for Table 2

c On reviewing this exception it was found that actually this claimant had died not of his lung cancer but of pneumonia following a cerebral vascular accident, or stroke.

a number of medical experts testified that the aneurysm was indeed a direct result of the accident and the claim for survivor's benefits was consequently allowed. And 5) Claim # 75, the case of a worker who had been receiving an 80% P.D. award for low back strain and died from an overdose of a drug which had been prescribed for the pain resulting from his compensable condition. This claim, too, was allowed.

It is of interest that a judgment as to the relationship between the worker's death and the compensable injury had to be made in 4 of the 5 cases; in 2 of these 4 the claim for survivor's benefits was indeed initially rejected and arguments certainly could have been made against allowing all 4 claims. These cases serve to illustrate the considerable subjective element involved.

The adjudication of claims for compensation by the survivors of workers who were receiving compensation for respiratory disease is often even more controversial, as will be discussed in Section 2. In this group cardio-respiratory deaths accounted for 32 of the 56 claims filed by survivors. The 3 allowed claims in the "other illness" and "unknown" columns were from the individuals who had received 100% P.D. awards. The 2 allowed fatal cancer claims were on behalf of an asbestotic and a silicotic. In neither case was the compensable respiratory disease responsible for the death, but in the former, the asbestos-exposure was deemed to have caused the cancer and in the latter, P.A.H. exposure in a foundry explained the allowance. (See Chapter II.)

Six of the 12 allowed claims by survivors of workers who died of respiratory disease and had been disabled by it prior to their deaths were on behalf of those who had received 100% P.D. awards. The other 6 allowed cases consisted of: a worker receiving a 25% award for silicosis who died of bronchopneumonia, a silicotic receiving a 30% award who died of advanced pulmonary fibrosis, two other silicotics both receiving 75% P.D. awards who died of "asphyxia" and "respiratory failure", another silicotic who died of respiratory failure but who had been receiving only a 60% P.D.award, and an asbestotic receiving a 60% P.D. award who died of respiratory failure. Several of these claims will be discussed in Section 2.1.

From reviewing these cases it was certainly not evident that they differed significantly from the 8 respiratory deaths that were not considered related to the compensable respiratory disease. These latter cases included 2 silicotics receiving 100% P.D. awards who died of pneumonia and respiratory failure respectively. In one case the WCB doctor attributed the claimant's death to respiratory pneumonitis secondary to alcoholism; in the second, the Board doctor felt that the claimant's prostate cancer was the predisposing cause to the respiratory failure, not the silicosis. The rejected claims for survivor's benefits also included the case of a silicotic receiving a 25% P.D. award who died of pneumonia but who also had leukemia, 3 silicotics receiving 30% awards who died of emphysema and bronchitis, anoxia and pneumonia respectively and 2 silicotics receiving 60% P.D. awards who both died of bronchopneumonia. Several of these cases will also be presented in Section 2.1.

The 7 cardiac deaths of workers who had received P.D. awards for respiratory disease between 20 and 99% were generally also contentious. Several such claims will be presented in Section 2.2 below.

It should also be noted from Table IV-6 that a claim for benefits by survivors of a worker with occupational cancer (assessed at 30% P.D.) was allowed despite the fact that the listed cause of death was coronary occlusion. (On careful review of the file it seemed that no determination to allow the claim for survivor's benefits had actually been made. In fact the circumstances surrounding the death had not yet been determined.) One rejected claim for benefits by the survivors of a firefighter who died of a heart attack 577 days after a compensable heart attack was also encountered. Several similar cases will be briefly presented in Section 3.4.2, as they, too, represent a contentious issue.

1.6 *Commentary*

As alluded to above, unless a claimant is receiving a 100% P.D. award at the time of death, or is deemed in retrospect to be likely to have been 100% impaired by his or her compensable disease, a judgement has to be made regarding the cause of death.

The dependent often finds it difficult to accept that the death was not at least partially caused by any disease that was serious enough to be found compensable, or that the worker's condition which ultimately proved fatal was not at least aggravated by his compensable condition. However, both in Peter Barth's review of asbestosis claims, and in the review of survivor's benefit claims for this Inquiry, it was noted that aggravation rarely seemed to be a significant factor in Board decisions regarding cause of death.

On reviewing the list of claims for survivor's benefits by specific compensable condition, compensation award and actual cause of death, in at least one-quarter of the claims it was certainly not obvious why the claim was either allowed or rejected. On personally reviewing most of these claims, this author found that many involved conflicting medical opinion, often even among the Board doctors themselves. Moreover, if the definition of "work-related" is stretched beyond that currently guiding the Board's practice, a good many more fall into this grey category. In fact it can be stated that only rarely does a disabled worker die of a cause completely and unequivocally unrelated to his or her compensable condition. Thus the complexities and uncertainties in evaluating the relative effect of one disease on another, and the extent to which any one particular disability contributes to a person's ultimate demise, indeed pose difficult problems for the compensation system.

Barth also noted the problems related to the inadequacy and inaccuracy of death certificate information, the fact that fewer autopsies are being performed in this province when compared to previous years and the difficulties encountered by the Board in receiving adequate information regarding the circumstances surrounding the death. He also alluded to the fact that when a worker's pension is terminated and the Workers' Compensation Board automatically considers the question of survivor's benefits, the dependents may not be made aware that the process is taking place and therefore may be unable to bring evidence. Unless specific initiatives are taken by the dependents, they will not even be informed of their right to appeal. As Barth found that this degree of initiative was the exception, this procedure was indicated as problematic.

The following sections elaborate on these issues and provide case examples to illustrate the complexities involved.

2. Controversy Over Cause of Death in Workers Who Were Disabled by Occupational Respiratory Diseases

2.1 Death from Respiratory Disease

All the fatalities from respiratory causes that occurred in 1980 among workers receiving permanent partial disability (P.P.D.) awards for respiratory disease were reviewed. Three cases from the 6 that were allowed as well as 3 from the 6 that were rejected are presented below:

Case 1 - Claim # 76 This claim for survivor's benefits was filed by the dependents of a 57-year-old miner who had been recommended for a 30% P.D. award for silicosis prior to his death. His "immediate cause" of death was listed on his death certificate as anoxia, with "morbid conditions giving rise to the immediate cause" listed as obstructive airways disease and pulmonary fibrosis. It was also noted that his "other morbid conditions" included cor pulmonale.

A compensation claim had first been filed for this worker's respiratory condition in April 1975. Although he had worked 25 years underground, including 8 years in the gold mines in the Kirkland Lake area and 7 years in the uranium mines of Elliot Lake, his claim was rejected by the Advisory Committee on Occupational Chest Diseases as there was no radiological evidence of silicosis.

In October 1979, the claim was re-established. The specialist from Kirkland Lake noted that the claimant was severely disabled with chronic obstructive lung disease, which completely disabled him from work and necessitated continuous oxygen at home. Highlighting the many years the claimant spent as an underground miner, he strongly urged that workers' compensation be granted. In November 1979 a Board physician reviewed the chest x-ray of 1975 and wrote: "I could see no real use at all today to follow this claim further because clearly this man's problem is emphysema and airways obstruction without any evidence at all of silicotic change. I would further think that the chance that the x-ray had changed in the last 4 years is about nil."

Meanwhile, medical reports supporting the claim were accumulating. The pulmonary function studies were felt by several respirologists to be compatible with pulmonary fibrosis as well as severe obstructive lung disease. One Board-employed physician who also served on the Advisory Committee on Chest Diseases noted that the changes on the chest x-ray were linear and streaky with no clear-cut discrete nodules in the lung field nor any suggestion of a conglomerate lesion suggestive of silicosis. Although alluding to the fact that the claimant had been a smoker of 10-12 cigarettes/day for more than 25 years before he quit in 1977, he felt that the claim merited reconsideration by the Advisory Committee in view of the claimant's 25 years of underground mining.

A referral to the Advisory Committee was made and a report finally received in April 1981. The diagnosis of silicosis was admitted as part of this man's problem in addition to obstructive airways disease and emphysema, and a retroactive award of 15% from January 1977 to December 1979 with an increase to 30% from January 1st, 1980 was recommended. Meanwhile, in July 1980, the claimant succumbed to his respiratory disease. Consideration for benefits to his survivors was consequently requested. In May 1981, a Board physician again recommended rejecting the claim, noting that the silicosis could not have played a significant role in the death. The Review Branch asked the medical department if there was any possibility that the deceased would have been assessed as 100% disabled just prior to death had he been re-examined. As the answer was negative the Claims Review Branch upheld the rejection. A request for an appeal hearing consequently ensued, and on last review was still pending.

Commentary: This rejected survivor's benefit claim resembled many others reviewed. The most striking factor here is that although the deceased had worked for 25 years in the dusty conditions of underground mining, the possibility of his chronic obstructive lung disease having been work-related was never considered by the Board doctor. In view of the many studies cited in Part A of Chapter III linking underground mining to chronic obstructive lung disease, including a study looking specifically at gold mines, and, more importantly, a study specifically linking mining in Elliot Lake with chronic obstructive lung disease, this lack of consideration is perplexing. Although the bitterness engendered by the 7 years of fighting this claim seemed to be largely unrelated to knowledge of the scientific justification for its allowance, the frustration and hardships ensuant upon rejection are all the more regrettable in appreciation of this evidence.

Case 2 - Claim 77 This claim for survivor's benefits was filed by the widow of an 80-year-old foundry worker who died of bronchopneumonia. He had been receiving a 25% P.D. award for what was listed as silicosis, however on careful review, the award was clearly granted for chronic obstructive lung disease.

The deceased had worked in an auto specialty foundry from 1936 to 1966 when he retired due to poor health. Although he had never smoked he had developed an irritating dry cough and breathlessness. When he was examined by the Advisory Committee on Occupational Chest Diseases in 1977 it was noted that he had marked obstructive lung disease with a slight restrictive ventilatory defect and suspected cor pulmonale. As there was no x-ray evidence of silicosis they recommended rejection.

The claim was appealed. The Appeals adjudicator asked if there was any industrial disease which could account for the obviously severe chest condition. In June 1979 a senior Board physician who specialized in respiratory disease cases, reviewed the claim and noted again that there was evidence of silicosis but that "the existence of severe airway obstruction in a non-smoker with a long history of foundry exposure gives food for thought..." He stressed, however, that "this rests on the assumption that he never smoked in his life." Moreover, he pointed out that "while emphysema and/or airways disease occurs primarily in smokers, possibly 15-20% of individuals effected have never smoked and never have been exposed to any occupational environment of significance." In recommending that the claim be allowed, he commented on the approach to the assessment:

> We do know that he was symptomatic by 1973 but capable of working in 1966. It would therefore seem reasonable to estimate that significant disability was present by 1970 assuming that it increased gradually to date to total disability. The average or mean disability over ten years would be approximately 50%. Taking into account the fact that he had T.B. and had part of the upper lobe removed, and considering the normal decrease in pulmonary function each year in the aging process, I would assign a 50% attribution factor to the mean disability since 1970 which would work out to be 25% per year commencing January 1, 1970.

The claimant died of pneumonia in 1980 with wheezing, extreme shortness of breath, gastrointestinal bleeding and cardiac arrhythmia prior to death. His post mortem showed widespread silicotic changes in the mediastinal lymph nodes which apparently caused an esophageal fistula, likely responsible for the gastrointestinal bleeding. The Board's consultant pathologist noted that while the lung parenchyma was quite free of silicosis, the silicotic changes in the hilar lymph nodes may have exerted some significant effect on pulmonary function. Noting too that there was significant bronchitis and emphysema in this non-smoking foundry worker, he recommended allowing the survivor's claim. Survivor's benefits consequently ensued.

Commentary - The allowance of this claim for survivor's benefits seemed to have been based on 2 factors: 1) that the effects of silica exposure were indeed found on post-mortem examination, and 2) that the deceased had been a non-smoker. First, the fact that the claim for silicosis had been rejected is worthy of note in this regard, highlighting the potential importance of post-mortem examinations in occupational disease claims. Secondly, in all the cases reviewed for this Inquiry this was one of only a few claims for chronic obstructive lung disease allowed outside the SO_2 guidelines. It was strongly suggested that had the claimant been even a light smoker the claim would have been rejected. This probably accounts for the rarity of allowed claims for chronic obstructive lung disease and again raises the issue of policy on this matter. Thirdly, the method of calculation of the P.D. award is worthy of note. Finally, with respect to the compensability of the fatality, this claim in juxtaposition with other claims reviewed, serves to illustrate the fine line between allowance and rejection.

Case 3 - Claim 78 This claim for survivor's benefits was filed by the dependents of a silicotic miner who had been receiving a 75% disability award for his respiratory impairment and died at the age of 44 from respiratory failure.

The claimant had worked underground in Elliot Lake from 1956 to 1967, then approximately 1 day/week underground and the rest of the time on the surface in a machine shop which he claimed was equally dusty. His claim for silicosis was originally allowed in 1967 at 25% P.D., and increased to 40% in 1975. He was examined by the Advisory Committee in 1980 at which time his chest x-ray and pulmonary function tests were reviewed and an ergonometric stress test was conducted. The Advisory Committee concluded that he had advanced silicosis and that he was incapable of performing any physical work. Noting that he smoked 20 cigarettes/day, a 75% P.D. award was recommended and he was scheduled for re-examination in 1 year's time.

Meanwhile, he was hospitalized in December 1980 with the diagnosis of unstable angina. It was noted, however, that the ECG revealed no significant ischemic changes nor anything suggestive of a myocardial infarction. As he seemed to be in no acute risk he was discharged on December 23 with a final diagnosis of unstable angina and a secondary diagnosis of "severe silicosis - complicating cardiac ischemia probably secondary to respiratory insufficiency." The claimant died on December 24.

The coroner's investigation concluded that the cause of death was asphyxiation (respiratory failure) and that the gross appearance of the lungs was probably due to silicosis. A medical consultant employed by the Board agreed that the death was compensable and survivor's benefits were allowed. The union representative requested that a retroactive increase in pension also be awarded to the widow as the deceased had been more than 75% disabled. In addition, the attending physician wrote to the Board stating that the claimant was 100% disabled prior to death. In response a Board physician pointed out that the Board deals with "industrial attributability not total disability". He noted that "the rather rapid downhill course this man experienced is certainly not typical of silicosis and the post-mortem showed that he in fact died of cor pulmonale or right heart failure secondary to pulmonary hypertension. Pulmonary hypertension itself is not a complication of silicosis..." In fact, he noted that "it might be argued that 75% was excessive." He suggested sending a copy of the autopsy report to the Advisory Committee for their opinion and that the Board's consulting pathologist should look at the slides as well. He noted that the man was quite young at his death and the thickening of the pulmonary arteries is rather unusual.

Accordingly, the pathologist examined autopsy material of the lungs. He noted that the claimant had pulmonary silicosis but "if the blocks available are representative, it is slight and unlikely to have caused serious dysfunction." He stated that he did not find vascular changes suggestive of pulmonary hypertension. He concluded that "there is nothing in the sections of lung to suggest how he died. If the blocks are representative," he noted, "I doubt if his silicosis contributed importantly to his death." On receipt of this report in February 1982, a senior Board-employed physician noted: "It is absolutely clear that the silicosis was mild to moderate only and that the 75% he was awarded was greatly in excess of that which one would expect from the tissue findings. Not only was our final award excessive, there seems to be no reason at all why we should consider that silicosis contributed in a significant way to his death." The adjudicator, noting the inconsistency between these recommendations and the post mortem report, requested a further opinion from the medical services division as to the probable cause of death. Offering no further explanation, the same Board physician noted that the Board would have been content to leave the claim as it stood had the union representative not attempted to secure the increased award for the widow. However, he noted "...in retrospect his death was probably primarily cardiac...At this time, technically, we have all the information we need to rescind the award to the widow if indeed such a procedure is within the present discretionary powers of the Board to implement." On final review, October 1982, the issue of rescinding the survivor's benefit award was still before the Claims Review Branch.

Commentary: This 44 year old miner with severe respiratory impairment had been receiving a 75% P.D. award when admitted to the hospital with non-specific cardio-respiratory symptoms. The coroner attributed his death to his compensable disease and the WCB had granted the survivor's benefit claim. Although the Board physician consulted in the case may have been right and non-occupational factors may have been underrated, there was no justification whatsoever to even contemplate rescinding the award. The extrapolation of a carefully qualified pathologist's report to starkly unequivocal conclusions is certainly not in keeping with the spirit of adjudication which the Act promises. This claim needs no further commentary other than to note the apparent inconsistency between the handling of this case and many of the others reviewed.

Case 4 - Claim 79 This claim for survivor's benefits was filed following the death of a 66-year-old smelter worker who had been receiving a 40% P.D. award for respiratory impairment and died in 1980 of pneumonia after a cerebral vascular accident (stroke).

In 1974 a Board-employed physician reviewing the case noted that the claimant was grossly overweight. He stated that:

> The pulmonary impairment is principally reflected by moderate to severe small airways disease which is almost certainly related to his 45 years of smoking and has nothing to do with any industrial chest condition that we can recognize. It is true that he did work in the Copper Cliff smelter since 1949 where he was exposed to various fumes and dusts but we have (not) yet of course recognized that the Copper Cliff smelter has produced an increased incidence of lower respiratory disease. It was acknowledged that he had lost some vital capacity from removal of the right upper lobe due to industrial lung cancer. He was thus estimated to be 40% impaired with half allocated to a pre-existing condition. The fatal claim was rejected.

Commentary: The circumstances of the death were not clearly described. For example, it was not stated how severe the stroke had been or how vigorously the pneumonia was treated. As the cause of death was considered respiratory in origin it may be assumed that the rejection of the claim was based on rejecting the occupational relationship of the underlying respiratory condition. A portion of the respiratory impairment was clearly occupational, that due to removal of a part of lung due to compensable lung cancer. Although, as discussed in Chapter III, the causal link between smelter exposure and chronic obstructive lung disease has now been acknowledged, it seems that the benefit of this decision was never applied to this case, either during the life of the claimant or with respect to the claim for survivor's benefits. This claim too, therefore, can be said to fall into the grey area of uncertainty.

Case 5 - Claim 80 This worker had been receiving a 60% P.D. award for silicosis. He was scheduled to be reassessed by the Advisory Committee in November 1980 but died in July 1980 of bronchopneumonia. On reviewing the file and clinical records at the time of death, a senior

Board physician noted that the pulmonary function studies showed relatively good lung function between 1967 and 1977 with the vital capacity having hardly changed at all and the FEV_1 having not dropped below 70%. He therefore concluded that this man would have been unlikely to have been 100% disabled from silicosis at the time of his death and therefore recommended rejection on this basis. The adjudicator, noting that silicosis and cancer of the pancreas were listed as contributory factors on the death certificate asked the medical services division if the death could have been related to the compensable condition. Again the response, this time from another Board physician was negative. The claim was rejected and, on last review, an appeal was still pending.

Case 6 - Claim 81 This 64-year-old miner had been seen by the Advisory Committee in November 1976, at which time his P.D. award for silicosis was increased from 25% to 75%. It was noted that he also had emphysema and chronic bronchitis. The worker was admitted to hospital in chronic respiratory failure which had been determined to be compensable prior to his death. He died while in hospital. No autopsy was carried out but a Board-employed doctor who reviewed the claim concluded that in view of the fact that the worker was receiving a 75% P.D. award, death benefits should be allowed. The survivor's benefit claim was thus allowed.

Commentary: These claims have been presented largely to illustrate the large element of subjective judgment and uncertainty that plagues the adjudication of claims of this nature. It would not be incorrect to conclude that respiratory impairment of any significant extent contributes to the ultimate demise of an individual who dies of respiratory disease. The approach described in the above cases has indeed met with a great deal of discontent.

2.2 *Cardiac Death Among Workers with Occupational Respiratory Disease*

The question of entitlement to survivor's benefits is also considerably problematic where the worker has a pulmonary impairment and dies from a subsequent cardiovascular condition. Barth observed, for example, that where a hypothetical worker, evaluated as 50% disabled from asbestosis, dies from a coronary condition it is likely that survivor's benefits would be denied. He noted that to many this seems harsh judgment because of the uncertainties regarding any linkage between the diseases. He did observe at least two instances in which the cause of death in asbestotics was diagnosed as cor pulmonale and as such was compensated.

The scientific literature on the relationship between cardiac and pulmonary disease was briefly alluded to in Part A of Chapter III. In recognition of this area of controversy, the WCB has indeed devoted considerable attention to this matter over the last 20 years. Prior to presenting the cases encountered it is worth summarizing these efforts and the findings that resulted.

By the end of the 1950's, the WCB had perceived the divergence of opinion with respect to the relationship between respiratory disease and cardiac death. Many claimants, their attending physicians and several experts seemed to feel that chronic oxygen deficiency resulting from severe lung disease might proceed to a point where coronary shutdown or myocardial infarction was precipitated and therefore myocardial infarction in silicotics should be compensable. Although this was physiologically quite plausible and a few reports had already appeared in the scientific literature attesting to the increased incidence of arrhythmias (disorders of the heart rhythm), there was no scientific evidence that atherosclerotic heart disease or cardiac mortality was increased in those suffering from silicosis, other pneumoconioses or even chronic obstructive lung disease.

In early 1960, a senior Board physician who specialized in chest disease cases wrote to an international expert on the subject, requesting his opinion. In a letter to the Board dated March 19th, 1960, the expert from Columbia University cited several studies and concluded that "myocardial infarction and/or coronary artery disease are not influenced by silicosis or even by right ventricular hypertrophy and dilatation." He also noted that even in very severe cases of secondary polycythemia (proliferation of red blood cells caused by oxygen deficiency), coronary thrombosis (the clumping of formed blood particles that obstructs blood flow in the blood vessels of the heart) is exceptionally unusual. He commented that "...in the presence of coronary disease in silicotics, the problem is not whether the latter favours the development of the former, but whether the manifestations (emphasis added) of coronary insufficiency are not, to some extent, more apt to become severe, if disturbed hemodynamics due to silicosis is present (reduction in cardiac output). I would give such patients the benefit of the doubt; however I would avoid correlation of development of coronary disease and infarction with cor pulmonale."

Accordingly, the Board's medical advisors operated on the premise that chronic chest disease and the respiratory imbalance that results does not cause coronary artery disease, coronary thrombosis or fatal myocardial infarction. Where no evidence exists of myocardial infarction or acute coronary thrombosis underlying a death in which the cardiovascular and respiratory elements are virtually indistinguishable, the Board states that the principle cause is assumed to be respiratory and the claim allowed.

In a letter to another cardiology specialist in December 1969, a senior Board doctor, wrote: "As medical advisors to the Board we are very willing to take a second look at our traditional approach to this matter if it can be shown that the cardio-respiratory physiology involved supports the previously mentioned anoxia (oxygen lack) theory as a major precipitating factor in coronary thrombosis or myocardial infarction." The reply he received was unequivocal: "The presence of atherosclerotic heart disease and the occurrence of myocardial infarction has nothing whatsoever to do with the fact that these patients have pneumoconioses." However, again to complicate the compensation question, this expert noted that patients who have chronic cor pulmonale have a more difficult post-myocardial infarction course. She pointed out that this was not because the hypoxia increases the incidence of either arrhythmias or myocardial infarction, which, she felt, does not happen, but because the hypoxia from the lung disease will be aggravated by the low cardiac output.

By the late 1970's a debate had been generated in Britain in reference to coronary artery disease and coalworkers' pneumoconiosis. The Ontario WCB medical advisors wrote to the Pneumoconiosis Medical Panel in London with regard to their conclusions concerning the scientific studies cited in the literature. Again, their conclusions were straightforward: There is no evidence that advanced chronic lung disease is associated with a higher than average death rate from coronary artery disease; hypoxia does not play a significant role in the causation of arrhythmias; chronic hypoxia due to chronic lung disease is unlikely to contribute to the development of myocardial infarction; airway obstruction is not correlated with an increased risk of coronary artery disease; moreover hypoxia, which may be present in severe lung disease, may cause dilation of the coronary arteries to increase, rather than decrease, blood flow in the heart; and finally, myocardial infarction may be significantly less common in the presence of chronic obstructive lung disease than in its absence.

The compensation struggles continued nonetheless. In September 1979, a University of Toronto pathologist, reviewed the literature for a second time at the Board's request and again concluded: "Pulmonary disease including silicosis does not increase the risk of developing myocardial infarction." But again that complicating factor was alluded to: "Whether silicosis increases the risk of dying of myocardial infarction once an infarct has developed is another question. Indeed, any severe disease reduces the likelihood of surviving a second severe disease. There can be no doubt that in this sense silicosis does increase the risk of dying from myocardial infarction once an infarct has developed." He did note, however, that this increase must be slight, as cardiac mortality has not been noted to be elevated in these workers.

Thus there seems to be a high degree of consensus that there is no correlation between respiratory disease and coronary artery disease. There still is considerable debate regarding the risk of cardiac arrest secondary to arrhythmias induced by hypoxia. However, perhaps most importantly, there seems to be a consensus that severe respiratory impairment increases the morbidity and mortality associated with myocardial infarction. The problem therefore seems to relate to the difficulty in quantifying the extent to which the compensable condition (in this case, respiratory) acted together with the non-compensable condition (the myocardial infarct) to produce a fatal outcome directly attributable to the latter. The decision as to how much indirect effect is necessary is a policy decision not a question of science. With this as background, the following cases illustrate the Board's current policy.

Case 1 - Claim 82 A survivor's benefit claim was established for dependents of a 72-year-old miner who was receiving a 30% permanent disability award for silicosis. His death was due to a myocardial infarction and it was contended that the compensable chest condition was an extra strain on the heart and therefore led to the death. Also the patient had been scheduled to undergo cardiac surgery, but doctors would not proceed due to the underlying lung condition. The autopsy report showed silicosis as a cause of death. The Board physician who reviewed the claim stated that heart disease was the cause of death, and the Board's pathologist stated that the patient had myocardial ischemia, chronic bronchitis, mild emphysema and that the silicosis was not significant enough to cause his right ventricular and pulmonary artery problems, which he felt were due to severe left ventricular failure and aortic stenosis. The appeal was denied.

Case 2 - Claim 83 A survivor's benefit claim was established following the death of a 76-year-old worker who had been receiving a 50% permanent disability award for silicotuberculosis, and died of heart failure. A causal relationship was contended as in the above case. He had not been seen by the Workers' Compensation Board for six years prior to his death, as in the words of the Advisory Committee on Chest Diseases, "this 70-year old claimant is too frail to report for examination in the future. We recommend that further listings be dispensed." Despite the fact that the patient had stated that he had been getting worse, the chest x-rays had shown no changes and hence there had been no increase in compensation at the last assessment. The claim for survivor's benefits was denied, as his request for an increase in permanent disability award had been prior to his death.

Case 3 - Claim 84 A claim for survivor's benefits was established in the death of an 80-year old silicotic who died of a myocardial infarction in October 1980. He had been receiving a 75% permanent disability award, but had not been assessed by the Advisory Committee on Chest Diseases since 1976. It was, however, deemed that he would probably have been 100% permanently disabled by the time of his death. It was noted that many years previously he had been put on digitalis by his doctor for right heart failure, secondary to pulmonary disease, but at the time the Board felt that the silicosis was relatively not disabling. The claim was nevertheless allowed.

Commentary: These three claims (derived from the Sample Survey of silicosis claims described in Chapter III-Part B) illustrate the controversy with respect to cardiac deaths in silicotics. In Case 1, supposedly cardiac surgery which may have helped to alleviate the underlying coronary condition was not performed due to the underlying lung condition. (The effect of compensable respiratory disease on fitness for surgery is also a major issue in the next case to be presented below.)

It may be noted that in Case 2, the worker had not been seen by the Advisory Committee of the Board for six years, but as his chest x-ray failed to show changes on his previous examinations, the assumption that his disability had stabilized at 50% was made and the claim was denied. The claimant in Case 3, however, was not seen by the Advisory Committee for three years and it was concluded that his 75% disability had likely increased to 100% by the time of his death. The fact that the patient had been treated for cor pulmonale was likely a strong factor in the allowance of this claim. (Although the above three claimants were all over 70 years old at the time of death, several claims were reviewed, specifically those of asbestotics, in which the claimant had pneumoconiosis and died of a cardiac cause at an earlier age.)

The approach used in British Columbia is interesting in this regard. As Barth observed, under Section 6(11) of its statute, the Workers' Compensation Board of that province must conclusively presume that the death of a worker with an industrial disease that impairs the capacity or function of the lungs, is a result of that disease where death is caused by an ailment of the heart or lungs. The presumption is limited to non-traumatic cases and the worker must have died prior to the age of 70. Barth points out that variations of this scheme could consist of altering the age limit or broadening or narrowing the terms of the diseases that would trigger the presumption. Some individuals and groups made presentations to the Weiler Inquiry suggesting the establishment of a schedule of diseases in this regard. Although the scientific evidence to support such a presumption is shaky, such a scheme may well be warranted purely on administrative and humanitarian grounds. (Another option listed by Barth was the linkage of the survivor's benefits to the size of the partial disability award regardless of cause, although as he points out, this would be inconsistent with the present approach and may be even more contentious.)

2.3 Other Causes of Death Among Workers with Industrial Lung Disease

The above two sections illustrated the uncertainty that often exists regarding the compensability of death in workers with industrial lung disease who die of pulmonary or cardiac causes. The following well-publicized case illustrates that in fact other circumstances of death of workers severely disabled by compensable lung diseases may spark considerable controversy.

Case 1 - Claim 85 This 54-year-old asbestos worker, employed for 22 years with a major asbestos manufacturer, submitted a claim for asbestosis in 1974. In May 1975, the Advisory Committee on Occupational Chest Diseases confirmed asbestosis and estimated the disability to be 25%. The claimant contested that his assessment was low. The Claims Review Branch denied the appeal, but granted a supplementary award of 75% to provide 100% benefits nonetheless. He was released in April 1976, when his disability assessment was increased to 40% and a special supplement of 60% was approved.

In January 1977, the claimant's treating internist suggested that he had severe asbestosis, was completely incapacitated and deserved full and permanent compensation. In March 1977, it was noted that aside from alcoholic hepatitis and asbestosis, the claimant had an anaplastic cancer of the thyroid, for which he subsequently received intensive radiation therapy. In July 1977, his treating internist indicated that his extensive pulmonary disease had led to cor pulmonale. It was noted that the cancer of the thyroid had responded poorly to radiotherapy. The claimant was hospitalized with respiratory disease and collapse of his left lung and died on July 27, 1978.

The attending physician at the time of death felt that the primary cause of death was asbestosis and that the cancer of the thyroid and complications of radiation therapy were secondary. The autopsy report noted the tumour of the thyroid gland which was necrotic with evidence of the radiation therapy, as well as fibrous plaques of the diaphragm, pleural adhesions, bilateral pleural effusions, pulmonary fibrosis, severe pulmonary edema, metastatic tumour of the right lung and biventricular cardiac enlargement. The summary stressed that the outstanding findings were those of pulmonary asbestosis and that the final episode was widespread pneumonia. The pathologist also noted that at the time the thyroid tumour was first diagnosed in December 1976, it was impossible to remove it surgically because of advanced pulmonary disease and that there was reasonable opinion that had surgery been performed at that time, a cure could have resulted.

A Board physician then telephoned the pathologist at the hospital and was told that the cause of death was attributed to the difficulty of breathing caused by the extensive reaction to the radiation therapy that the worker received for his thyroid cancer. The Board's pathologist thought that terminally the worker had suffered from pulmonary edema, which was in part due to his heart failure and in part, due to his pneumonia. A surgeon consulted by the Board noted that this type of cancer of the thyroid was very malignant and only rarely amenable to curative surgery. The Industrial Chest Disease Service of the Ministry of Labour felt that the worker died of complications of his thyroid cancer as well as heart failure and terminal pneumonia. It was felt that his respiratory status as late as May 1977 would not have precluded any necessary operative procedure.

Having examined all the medical evidence on record, it was the view of a senior WCB physician who specialized in respiratory disease claims and consistent with the advice of the Advisory Committee that the worker died from complications of thyroid cancer. It was thus concluded that there was no evidence upon which the Board could justify accepting a claim for survivor's benefits on the basis that the worker's death was due directly or indirectly to asbestosis, and in January 1979, the claim was denied.

This was not at all satisfactory to the widow and an appeal ensued. The claimant's family practitioner and the internist came to give direct evidence at the appeal hearing and reiterated, among other things, that surgery had not been performed due to the presence of asbestosis. No other medical practitioners were present at the hearing.

Following the appeal hearing, a letter was sent to the radiotherapist who had participated in the decision not to perform surgery asking him to confirm his statements and to provide a curriculum vitae. A transcript of the appeal hearing was distributed to senior members of the Board's medical services division to evaluate the testimony of the two medical witnesses and to express an opinion about their credibility. In their responses, the Board doctors presented arguments supporting the denial of the claim, and seriously questioning the competence and credibility of the physicians who testified. It was insinuated that there was an attempt to deliberately mislead the panel as no pre-operative assessment was even performed regarding the patient's suitability for surgery.

Meanwhile, the widow had sent letters to the Ministry of Labour and to the Chairman of the Workers' Compensation Board to enlist their support. Counsel for the Energy and Atomic Workers Union, acting on behalf of the claimant, asserted at the time that "the whole affair has degenerated into a test of the relative credibility and competency of medical practitioners on both sides." On August 13, 1980, the Appeal Board announced its decision to deny the claim. However, on August 22, 1980, under the provisions of Section 75, the decision of the Appeal Board was reconsidered: citing now that the reason surgery was not done was due to asbestosis, the claim was finally allowed and the employer was given 80% relief from the costs of the survivor's benefit claim.

Commentary: This case again illustrates the extreme importance of the percentage of the worker's disability award prior to his death, had he been assessed as 100% permanent disabled - as had been persistently requested: rather than 40% disabled with a 60% supplement, the survivor's benefit claim would automatically have been allowed.

Secondly, this case highlights the extreme importance placed on medical opinion and particularly on the advice of WCB doctors. Over two dozen medical opinions were expressed during the course of this case, over half of which were from Board doctors or independent consultants directly solicited by the Board. The remainder were from physicians directly involved in the case. The extent of the claimant's asbestosis was debated; the malignancy and potential surgical cure of the thyroid cancer, the possibility that the thyroid cancer had been mismanaged from the start, the possible suitability of the claimant for surgery, and ultimately, even the cause of death was disputed. (This commentary will not add yet another medical opinion to the debate, other than to note the studies referred to in part A of Chapter III asserting an unequivocally increased mortality among certified asbestotics, particularly the increased mortality from non-malignant respiratory causes, as well as from malignant disease.) Not only were the specific medical hypotheses evaluated, but the competence, credibility and integrity of the individual physicians holding views contrary to those of the Board were put into question.

Finally, if the Board's decision was that this man's death was due to surgery having been denied as a result of his asbestosis, one wonders if it was indeed appropriate for the company to be given 80% relief. This is very unusual in survivors' claims, and perhaps is evidence of the need for an expanded fund for the many other cases of this nature, which, for the most part, are presently merely denied.

Thus the above three sections illustrate the extremely contentious nature of survivors' claims resulting from deaths of workers who had been suffering from occupational respiratory diseases, whether death is ultimately attributed to respiratory, cardiac or other causes.

3. The Compensation of Cardiac Disease

3.1 Statistics

The number of cardiac claims processed by the Ontario Workers' Compensation Board is minute in comparison to its total volume (although it is among the highest in all Canadian jurisdictions). For example in 1974, of the 443,000 claims allowed, a mere 43 were entitlements for a cardiac disability. While only 180 cardiac claims were even received that year, the rejection rate of 76% is quite telling of the difference between these claims and the usual accident claims. The numbers have remained relatively constant over the years. In 1969, 201 cardiac claims were submitted, of which 41 were allowed. In 1979, of 296 claims submitted, 59 were allowed, again for a rejection rate of 80%.

The total number of survivor's benefit claims assessed in 1980 was 579, of which 324 were allowed. (See Table IIB-2a in Chapter II Part B). Of the 579, 273 had listed as the cause of death: accident, trauma, electrocution, burn, asphyxiation, collapse, motor vehicle accident, murder or some other traumatic cause; in 243 the cause of death was listed as a disease. In this group, 82 were for cardiac deaths. These figures of the claims reported in 1980 reveal the same pattern as the figures presented in Section 1 of this chapter regarding deaths that occurred in 1980. From either source it can be seen that while cardiac claims represent a tiny portion of the total claims, they represent a significant portion of claims for survivor's benefits.

Of the 42 "old" cardiac claims on behalf of survivors, 14 were allowed and 28 were rejected. In some of these, the allowed claim was for a previous cardiac episode, from which the claimant never fully recovered. In others, compensation was for another condition, alleged to have played a contributory role in the final demise. Those in which the antecedent condition was respiratory have been alluded to above; several cases of cardiac death in the presence of other compensable conditions will be presented in Section 3.4.

Of 39 "new" claims filed by survivors of workers who died of cardiac causes (excluding trauma, electrocution and the like), 4 were allowed. (Likely, had the traumatic claims been reviewed, more "cardiac" deaths would have been uncovered.) Regarding the exceedingly high rejection rate of 89.7%, it should be noted that many of the new claims were established merely because the individual was at work at the time of the myocardial infarction, without anyone necessarily contending that the workplace played a causal role in the event. According to the Act, death on the job is to be presumed work-related unless the contrary is shown.

3.2 Policy

Atherosclerotic heart disease is not considered to be an industrial disease under the terms of the Ontario Workers' Compensation Act. In cases involving direct trauma or electrical injuries involving the heart, the causal agent is obvious and entitlement follows. Claims, however, are not accepted where a heart condition is due to general overwork, physical exhaustion, emotional strain, prolonged nervous tension or long hours at work unless there is a work incident or other direct causal relationship. Policy stipulates that exertion unusual for a particular worker must have preceded the attack in order to entitle the claimant to compensation. However, claims involving severe physical exertion, lack of oxygen, smoke inhalation and various noxious gases and fumes, acting singly or in combination, may be considered on their own merit.

When permanent impairment of cardiac function results, the pension awarded is the full amount of the total clinical rating (and a portion, usually 50%, is charged to the Second Injury and Enhancement Fund). However, as the claim is allowed on an aggravation basis, the claimant is denied entitlement for any further attacks. Subsequent heart attacks are not accepted unless there is "another industrial occurrence" which would create entitlement for a new claim. When a worker receiving a pension for a heart condition dies as a result of a further cardiac disturbance, the fatal claim is not accepted, irrespective of the amount of the permanent disability award, unless death is prior to recuperation from the original attack. (This would include most claimants who had received entitlement for cardiac disability, but had not yet been assessed for a permanent disability award which usually takes place approximately one year after the initial event.) Several cases will be presented to illustrate this policy.

A second group of cardiac disability claims allowed are those secondary to complications of other compensable diseases or cardiac complications which occur during treatment, specifically during anesthesia or during the immediate post-operative period. Several case presentations of this policy have already been presented, and several further cases will be discussed below.

A complete outline of the adjudication guidelines is attached in Appendix 1.

3.3 *Evolution of Guidelines*

Cardiac disability has been compensable in Ontario for many years. In 1958, at the request of the Medical Aid Committee, a prominent cardiologist was solicited regarding his opinion concerning medical aid for claimants awarded a permanent disability pension for cardiac disability. Based on his recommendations, it was decided to disallow medical aid following establishment of the P.D. award unless a new claim was established, with exceptions only at the recommendation of a cardiologist who "upon establishing an accurate and precise diagnosis defines the degree and duration of liability to be assumed by the Workmen's Compensation Board."

The matter was reviewed in 1967. The adjudication of heart claims in other jurisdictions was carefully assessed by the Board. Also, submissions had been presented to The Honourable Justice McGillvary, who was conducting a Royal Commission into matters concerning workers' compensation in Ontario at that time. The Provincial Federation of Ontario Professional Firefighters, for example, contended that the duties of firefighters resulted in an increased number of cases of injury or death from arterial hypertension, heart or respiratory disease. They pointed out that legislation covering firefighters existed in some of the states of the U.S. and in Manitoba in 1966 a regulation was made stating that:

> ...where a firefighter suffers a heart attack...while he is still engaged in the actual answering of a fire call and the fighting of fire; or while he is actually engaged upon prescribed training phases of his work involving a substantial physical, mental or nervous tension or strain, and...unless the contrary is proved, the injury shall be deemed to have been suffered in the line of duty and to have arisen out of and in the course of his employment.

The Federation admitted that those claims that had been rejected by the Ontario Workers' Compensation Board were not a result of fighting particular fires, but by reason of heart or lung disease which the Federation stated should be related and can be related to this occupation. Justice McGillvary noted that this submission received support from many labour groups including the United Steelworkers of America and the United Electrical Workers, the latter extending its submission to ask for greater flexibility in the adjudication of all heart claims. He concluded that he would be prepared to make a recommendation similar to that of the Manitoba regulation "but as no difficulty is experienced at the present time in the manner in which the Board deals with heart attacks suffered in the course of firefighting, such a regulation hardly seems necessary." He expressed approval of the manner in which the Board handles heart claims "so long as they receive careful consideration and are judged on their merits with the benefit of the doubt being awarded to the claimant no more can be demanded in a workmen's compensation case."

The Board reviewed and reiterated its guidelines in 1977. In 1979, trends developing in other jurisdictions were again assessed and a literature review was conducted which included topics of effort-induced as well as emotional causes of sudden death; the immediate antecedents of myocardial infarction were explored, as well as the effects of life changes, including illness and disability, on the onset of illnesses. It was pointed out that Ontario is more lenient than other jurisdictions in this area and it was not considered that there had been sufficient evidence produced in the literature review to warrant expansion of benefits, for example, in the area of emotional stress. It was noted that this area would be increasingly explored over the next decade or so.

The subject was also discussed at the meeting of chief medical officers during the Conference of the Association of Workmen's Compensation Boards of Canada held in Vancouver in June, 1979. Subsequently, a series of joint meetings was held by the Medical Services Divisions and the Claims Services Divisions which resulted in the proposed guidelines. The comment was made that "the concept that a causal relationship shall be considered to be in effect until proven otherwise is not acceptable from the medical standpoint. A causal relationship is not considered established until it has been shown to exist scientifically."

3.4 Case Presentations

3.4.1 Heart Attacks on the Job

As noted in the Adjudication Manual, the claims are categorized into two groups: where the causal agent is obvious and demonstrable (including traumatic injury to the chest and electric shock), and where the causal agent is in doubt (including stress or strain). All survivor's benefit claims in the latter category that were set up in 1980 were reviewed, some of which are presented below. Allowed claims are over-represented in this sample in order to adequately illustrate the principles on which entitlement is based.

Case 1 - Claim 86 A 47-year-old truck driver suffered a fatal myocardial infarction while repairing a broken plank on the box of his dump truck. The plank was 2" x 8", approximately 12 feet long and weighed an estimated 50 lbs. The Board doctors felt that "unusual stress is involved in replacing wooden planks" and therefore recommended allowing the claim. Interestingly, the adjudicator, who apparently had some experience as a truck driver, stated that truck drivers have more strenuous jobs than the above. Feeling that this stress was not "unusual" he recommended rejection. The section director intervened to recommend allowance and the claim was thus allowed.

Case 2. - Claim 87 A 46-year-old steelworker suffered a fatal myocardial infarction while working around a steel furnace, admitted by the employer to be an extremely hot area. The union representative stated that the worker was swinging a sledge hammer and was exerting extra physical effort in performing his job. It was noted that he had no past history of heart disease and no predisposing risk factors were listed. The claim was, nonetheless, rejected.

Case 3. - Claim 88 A 48-year-old roads superintendent was shoveling hard ground which was stated "not to be an unusual part of his work." He was not symptomatic at the time of shoveling, however, hours later he suffered a myocardial infarction. Seven months later he suffered a second fatal myocardial infarction. The Board doctors felt that "shoveling gravel can be quite heavy strenuous work, so it is possible that the claim has merit." As it was also felt that he had not recovered from the October 1979 episode, his death in January 1980 was deemed compensable.

Case 4. - Claim 89 A 43-year-old labourer suffered a myocardial infarction two days after developing chest pain while performing heavy and strenuous tasks associated with sandblasting, and use of heavy equipment and heavy construction vehicles. Two months later he suffered his second, this time fatal, myocardial infarction. It was noted that he had no past history of heart disease prior to this event, although he was moderatly hypertensive and obese. The Board doctor felt that "the question is whether it should be considered as part of his regular duties." As he had been doing this particular work only one week, the claim was allowed.

Case 5. - Claim 90 A 50-year-old seasonal painter suffered a myocardial infarction within three days of his return to work to begin the season. It was noted that he had had a previous infarction years earlier and that he was obese and had chronic bronchitis. The claim was rejected.

Case 6 - Claim 91 A 59-year-old plumber (foreman) was doing a job usually done by labourers due to the shortage of help. While using "equipment he had not used in years" he suffered a myocardial infarction. The Board doctors felt that this was "unusual and unaccustomed strain." They recommended that the claim be allowed "on the basis of aggravation of underlying atherosclerotic heart disease." His condition stabilized and he returned to work in May 1980, four months after his initial attack. He was scheduled to return in December for a cardiology appraisal regarding a permanent disability rating, however, in June of that year he suffered another myocardial infarction and died. As permanent disability had not yet been established, the claim for survivor's benefits was allowed without further investigation.

Case 7 - Claim 92 A 56-year-old labourer in a liquor store was lifting a case of whiskey estimated to weigh 40 to 50 lbs. when he experienced chest pains, shortness of breath, and a feeling of compression in his chest. The diagnosis of this event was not confirmed as no electrocardiograph changes were documented. Six weeks later, he suffered a fatal myocardial infarction. It was noted that the worker was hypertensive, diabetic, was a smoker, and had a history of gout and alcohol consumption. Claims for both the initial and fatal events were rejected as being due to underlying atherosclerotic heart disease.

Case 8 - Claim 93 A 72-year-old warehouseman had been lifting boxes of canned groceries for approximately an hour when he suffered a fatal myocardial infarction. Three months previously, he had an illness diagnosed as myocarditis (inflammation of the heart), but no past history of atherosclerotic heart disease nor other relevant risk factors. The claim was denied.

Case 9 - Claim 94 A 62-year-old cameraman suffered a myocardial infarction while filming at the office of the Mayor. Six weeks later, he suffered a second and fatal myocardial infarction. His wife stated that "he was under a great deal of emotional stress, he was overworked, had to lug around heavy equipment and he was under constant pressure due to his irregular schedule." As this was deemed to be all part of his job, the claim was rejected.

Case 10 - Claim 95 A 42-year-old police officer suffered a fatal myocardial infarction while arresting a suspect. There was a question as to some slight struggle which may have taken place while the officer attempted to alter the suspect's handcuffs. It was noted that "he was upset and emotional while conversing but was not struck or knocked down." The officer's widow felt that "the cause of her husband's heart attack was the constant pressure and stress of his job as a police officer." He had no past history of heart disease and there was no mention made of lifestyle risk factors. The adjudicator felt that as there was nothing unusual in this event sufficient to cause a heart attack the claim should be denied. One of the Board doctors agreed to deny, unless evidence of violence or unusual activity could be found. The autopsy showed only "an abrasion on the left thigh". A second Board doctor was "inclined to give the benefit of the doubt" and the claim was allowed.

Commentary: Policy regarding entitlement, as outlined in Section 3.2, is that emotional strain, nervous tension, general overwork, physical exhaustion or long hours, are not relevant factors. Thus, the task of the medical consultant is to ensure that the diagnosis has been established and to decide whether the physical strain of the job could be considered "unusual or excessive". In four of the five allowed claims described above, entitlement was granted on the basis of the work being other than the claimant's "usual" job; in one case (Case 3) although it was noted that the claimant was performing his usual work, the medical consultants felt that the job was excessively strenuous. In two other of the allowed cases, (Case 1 and Case 10), there was some question as to how "unusual" or "excessive" the event in question really was and the "benefit of the doubt" prevailed. In two others (Cases 5 and 6), it was well established that the task was not routine for the individual in question. In one of the rejected claims, however (Case 5), the work was also not in the individual's established routine (i.e. only three days on the job, as opposed to one week in the allowed Case 4), and in at least one other rejected claim (Case 2) one wonders why it was decided that excessive demands were not being made (i.e. excessive exertion in an extremely hot environment).

Although much attention is devoted to the assessment of the medical history of the individual (past medical history, lifestyle risk factors, ECG, lab reports) much less attention seems to go into the assessment of the demands of the job. It seems that the investigation often amounts to a few interviews to obtain a vague job description and ascertain if the work on the day in question was "unusual". Certainly, if more in-depth assessment of the workplace is indeed carried out, no detail is found in the claim file. (For example, the temperature, ventilation of the workplace, amount of energy expenditure the task requires, posture the worker has to assume, the sequence of movement, the weight and design of the materials and machinery, let alone the non-physical stress factors, are not commented upon). Occasionally, only the conclusion that "there was nothing unusual" is reported. Apparently industrial hygiene consultations are not solicited in these cases. Certainly no ergonomic assessments are carried out (i.e. the suitability of the job to the worker in question). For example, lifting 50-lb. boxes may indeed have been excessive for the specific workers in Cases 7 and 8. Again, little information was provided as to how much carrying, bending and rotation was actually involved. It is possible that all these factors are taken into consideration by the investigator and subsequently by the medical consultant; nevertheless, the lack of detailed industrial hygiene documentation is notable.

Questions also arise concerning the objectivity of decision-making. For example, in Case 10, the bruise on the officer's thigh as evidence of excessive force was given as the reason for the allowance of the claim, although the explanation given by the officer's widow is at least equally as medically plausible (emotional stress, etc.). Interestingly, the widow in Case 9 put forward a similar explanation for her husband's death, however the policy decision on these matters clearly pointed to a rejection in her case.

The existence of previous cardiac disease does not seem to play a decisive role in these decisions. In some cases in which the claimants had documented past histories of heart disease the claims were allowed, whereas in other cases in which the claimants had had no documented history, the claims were rejected. The explanation for this lies in the accepted medical doctrine as discussed above, that coronary episodes do not occur without pre-existing pathology, and even in the allowed claims, the entitlement is based on "aggravation of pre-existing condition" hence explaining the provision of relief to employers from the Second Injury Enhancement Fund. Case 3 above demonstrates the policy of allowing a fatal cardiac claim if it occurs prior to recuperation from a compensable myocardial infarction. The following case provides an illustrative contrast:

Case 11 - Claim 96 A survivor's benefit claim was established following the death of a 63-year-old worker from a myocardial infarction in May 1980 while he was receiving total temporary benefits for unstable angina which resulted from having exerted himself at work in December 1979. ("Unstable angina" is basically a condition of insufficient blood flow to the heart, but which has not actually caused the infarction or death of heart muscle.) The claimant had had recurrent episodes of angina since the time of the compensable episode. The claimant's doctor felt that his fatal myocardial infarction was related to the compensable incident. The Board's cardiology consultant stated that atherosclerotic heart disease was likely well established before either of these dates. The Board denied that the exertion of December 1979 was responsible for "the inevitable progress of atherosclerotic heart disease." Thus, essentially the first claim was allowed on the basis of aggravation of pre-existing atherosclerotic heart disease, however, as death was due to the pre-existing condition, not to the aggravation thereof, the claim for survivor's benefits was rejected.

This case illustrates that the policy of allowing claims for survivor's benefits if the worker dies prior to his recuperation from a compensable myocardial infarction, is not extrapolated to the allowance of claims prior to recuperation from unstable angina. Although quite understandable from a pathophysiological viewpoint, it is not surprising that the difference between these situations is not immediately evident to claimants.

3.4.2 Cardiac Claims for Survivor's Benefits from the Survivors of Firefighters

Although there is still considerable controversy in the scientific literature, some studies have suggested that firefighters are at increased risk of developing cardiovascular disease and, consequently, it has been suggested that special provisions be established to provide a presumption that heart disease in firefighters be considered work-related. The following is an account of the survivors' claims in cases of cardiac fatality received by the Workers' Compensation Board of Ontario in 1980 for firefighters.

Case 1 - Claim 97 A 53-year-old resource technician was found dead at the fire line about to embark on fighting a bush fire. The cause of death was determined to be a myocardial infarction. Although he had not yet been exposed to the actual fire, the terrain was rugged and the strenuous exertion of the work was deemed to be contributory. The claim was allowed.

Case 2 - Claim 98 A 56-year-old volunteer fireman suffered a myocardial infarction in the fire station as he was about to answer a fire call. He had just telephoned his wife to inform her of the location of the fire. It was noted that he had had a history of angina and two previous heart attacks and that besides his work as a volunteer firefighter he had worked in the metal industry. The claim was rejected.

Case 3 - Claim 99 A 42-year-old firefighter suffered a fatal myocardial infarction while working a second shift as a firefighter. It was deemed that no unusual stress was placed on him at the time of his death. The claim was rejected.

Case 4 - Claim 100 A 59-year-old firefighter died of a myocardial infarction in May of 1980. He began work as a firefighter in 1946, and as stated by the Ontario Professional Firefighters Association, he had no evidence of heart or lung disease at the time. In 1966, he received compensation for heat exhaustion, and in 1969, he was entitled to benefits as a result of having inhaled aerosol vapours from a fire in a beauty station which caused ulcers on his eyelids. In 1977, it was recorded that he had had considerable exposure to polyvinyl chlorides (PVCs) in a fire, but did not require compensation as no medical aid was sought. (Later his union submitted various journal articles to the Board attesting to the cardiotoxicity of PVCs in presenting the claim for survivor's benefits following his fatal cardiac episode.) In 1978, he suffered a myocardial infarction, which was deemed compensable. After that time he was limited to light duties, but still maintained his job with the fire department so as to be eligible for completion of his 35 years of service pension. In May 1980 he suffered a second and fatal myocardial infarction. His union attested that his exposure as a firefighter (particularly his PVC exposure episode) contributed to his demise. The claimant's medical consultant contended that "his myocardial infarction in August, 1978, definitely shortened his lifespan." The Board doctors and the cardiologist consulted by the Board felt that he was not subject to undue strain after recovering from the 1978 episode, that the sequence of events followed a natural course for coronary atherosclerosis and that this was not attributable to the initial episode. The appeal was thus denied.

Commentary (Two further claims from firefighters who died of cardiac causes while on compensation for cardiac disease were encountered in the claims review of deaths that occurred in 1980. Both were rejected but as they were similar to case 4 they have not been presented.) From these cases it appears that the fact that a worker was employed as a firefighter did not significantly influence the claim. Policies and procedures that were in operation for other occupational groups seemed to prevail for firefighters as well.

3.4.3 *Cardiac Deaths and Workers Receiving Benefits for Compensable Illnesses*

Having alluded to the complexity of the pathophysiology of atherosclerotic heart disease in Appendix 6 of Chapter I, Part B, it should be easily appreciated that the adjudication of cardiac claims on behalf of workers who had been receiving compensation for a pre-established impairment, is a complex and difficult task. In the group of claims described in Subsection 3.4.1 and 3.4.2 , the issues revolved around the interplay of the work environment and the cardiovascular system of the individual. In this group, the issues revolve around the interplay of one impairment in an individual's internal functioning and another impairment or series of impairments. Clearly, all aspects of human biology are interrelated, hence the assessment from a physiological point of view is the significance of the specific interrelationships. Often the underlying issue for compensation purposes does not seem to be the relationship between the impairments, but the extent of the disability imposed by the compensable impairment, as already repeatedly illustrated. The following case provides yet a further example, and somewhat typifies the issues involved:

Case 1 - Claim 101 A claim for survivor's benefits was established following the death of a 56-year-old worker from a myocardial infarction in June 1980. It was contended that an injury of September 1978, for which he was still receiving total temporary benefits, contributed to the claimant's ultimate demise. The claimant's entitlements were listed as follows: post traumatic cellulitis, aorto-bifemoral by-pass graft, ulcer left leg and skin grafting. The list of non-entitlements included: diabetes mellitus, peripheral vascular disease, left common iliac occlusion, right iliac stenosis, bilateral femoral stenosis, atherosclerotic heart disease, myocardial infarction, lumbar bilateral sympathectamy, and the acute fatal myocardial infarction. The argument was made that there was a physiological relationship between the entitlements and the non-entitlements and the family doctor felt that the frustration, discouragement and apprehension resulting from the non-healing ulcer contributed to the sudden death. However, the claim that the myocardial infarction was significantly related to the entitlements was rejected. A possible appeal was still pending on last review.

Commentary The above case as well as those discussed in Section 2 illustrates the immense importance placed on quantitative disability assessments and type of compensation award. Had the claimant been receiving a 100% PD award instead of a TT award survivor's benefits would have been allowed. The contentious issue of the interaction of the underlying impairment and the compensable conditions in terms of the latter "aggravating" the effects of the former, or the "enhancement" effect of the interaction of the impairments was again raised. The Board has been repeatedly criticized for failing to take "aggravating effects" fully into consideration; certainly the task is not an easy one.

4. The Issues Summarized

1. Industrial fatalities in Ontario encompass a good deal more than fatal accidents that occur on the job. In more than 1/3 of the fatalities compensated in 1980 the cause of death was a disease. In assessing the impact of occupational factors on mortality it is therefore important to go beyond enumerating fatal accidents.

2. In 1980, survivor's benefits were awarded to the dependents of 60 workers who had been receiving 100% permanent disability awards. Although the cause of death was deemed by the WCB to be unrelated to the compensable condition in 26 of these 60, it seems that only rarely was the death very clearly unrelated. The automatic awarding of survivor's benefits in these cases therefore does not appear to be inappropriate.

3. The number of fatal claims that had to be adjudicated for cases of individuals who had been receiving permanent partial disability (PPD) awards in 1980 was 68. Of these only 25 originated from industrial

accidents, and 43 were for workers receiving PPD awards for industrial diseases. The bulk of these claims (36) were for workers with industrial lung disease, the majority of which (26) were rejected. These claims are the source of considerable controversy, often involving conflicting medical opinion and repeated appeals.

Thus, if a presumption had been established to allow survivor's benefits in all cases where the deceased had been receiving an award for permanent impairment attributable to industrial lung disease regardless of cause of death, the outcome of only 26 claims would have been altered. A presumption allowing all fatal claims where a worker with industrial lung disease dies of cardiac or pulmonary causes (as is the case in British Columbia) would have altered the outcome of only 13 claims.

(It may be argued that if there was indeed a presumption of this nature more claims for survivor's benefits would be filed and the actual number of fatal cases compensated would be much higher. This concern, however, is largely unfounded in that the assessment for survivor's benefits is already essentially automatic, as the Industrial Disease and Dependents Section adjudicators routinely submit these claims for evaluation. Moreover, the total number of cases of respiratory disease compensated in 1980 is not very large. As was shown in Table IIIB-3 of Chapter III, Part B, only 45 cases of silicosis were compensated, and 10 cases of asbestosis. The total number of workers compensated for bronchitis, and emphysema was 7; 21 asthma claims were compensated and the total number of "other pneumoconioses" (including conditions due to chemical fumes) was only 49. These two latter categories likely consisted largely of "TT only" claims and would be expected to usually result in full recovery. Thus the total number of cases of silicosis, asbestosis and chronic obstructive lung disease (62) approximately equals the current number of claims for survivor's benefits from dependents of workers who received compensation for these ailments (58). Given the slight variation from year to year, this can be considered as confirmation that a presumption allowing survivor's benefits in these cases could not possibly significantly increase the number of such claims filed.)

Given the uncertainty in the medical-scientific community in this area, the great deal of resources currently expended in the investigation and adjudication of this small group of claims, the long delays, and the extent of hardship, frustration and embitterment associated with these controversial cases, it would seem eminently reasonable to invoke a presumption facilitating the swift allowance of these claims, accepting the fact that in several cases possible "over-compensation" may indeed result.

Of the claims by survivors of workers disabled from industrial diseases other than respiratory (totalling about 40, including the "T.T. only" claims), only 5 had not yet resulted in the payment of survivor's benefits at the time of this survey (and only 1 of which was for a worker receiving a PD award). Although the establishment of a presumption (or series of presumptions) to govern the adjudication of these claims does not seem to be necessary, since its creation would certainly not change the outcome of a large number of claims, the formulation of presumptions in this group should be considered for the sake of expediting the one or two contentious claims which do occasionally crop-up (e.g. cardiac death in workers receiving compensation for cardiac disease). This handful of claims is invariably associated with the expenditure of a great deal of time, effort, cost and emotional energy of a large number of individuals. While perhaps not scientifically rigorous, the decision to provide "the benefit of the doubt" in these few cases could certainly not be considered completely unfounded.

In fact, the data indicate that there are very few claims in which the cause of death in workers with industrial diseases was clearly unrelated to the compensable condition. For example, none of the claimants disabled by industrial disease died of traumatic causes, and, in fact, the vast majority of claimants died of diseases of the same organ system as their compensable impairment. The provision of survivor's benefits to the dependents of all these workers would be neither frivolous nor grossly inappropriate.

4. The number of "new" fatal claims for workers who die of diseases is potentially much greater than the number currently being reported. The problems in adjudicating these claims relate to the difficulties in defining and determining the "work-relatedness" of the deaths as discussed at length in the previous three chapters.

   Section 3 of this chapter illustrated the specific issues with respect to assessing the work-relatedness of cardiac deaths. It was noted that the evaluation of the workplace or even the interaction of the individual worker with the workplace is greatly overshadowed by the assessment of the medical history of the individual in isolation. Often, it seemed, the issue was not whether the working conditions could have contributed to the worker's demise but rather could the terminal event have taken place even without the influence of adverse occupational factors. The necessity for the working conditions to be "unusual" for the individual in question was noted. Consideration should be given to the appropriateness of these requirements with respect to the purpose of compensation. These, clearly, are policy issues not answerable by the medico-scientific literature.

   Again, the formulation of certain presumptions to facilitate the swift allowance of cardiac claims under certain circumstances may be desirable.

5. It is evident that there is no widely-accepted clear-cut distinction between deaths that are "significantly" related to working conditions or compensable impairments and those which are not. Ultimately, the only satisfactory solution lies in a system where entitlement to financial support will not have to depend on which side of this fine line a case falls.

APPENDIX 1

Cardiac Claims - Guidelines For Adjudication

1. Entitlement for Cardiac Conditions

1.1 Cardiac claims are accepted as work related when:

1.1.1 a causal relationship is shown between the cardiac condition and an accident at work, or

1.1.2 the cardiac condition is established as a disablement arising out of and in the course of the employment.

Section 1(1)(a) of the Workmen's Compensation Act is the authority for acceptance of these claims.

1.2 Entitlement for a cardiac condition is considered under any of the following circumstances.

1.2.1 Traumatic injury, involving penetrating and non-penetrating injury to the chest wall.

1.2.2 Electric shock producing irregular cardiac rhythm.

1.2.3 Inhalation of smoke and various noxious gases and fumes (e.g. firefighters).

1.2.4 Complication of treatment for a compensable injury (e.g. anaesthesia with an interval of hypotension, hypoxia or cardiac arrest).

1.2.5 Unusual physical exertion for the individual and/or acute emotional stress with no significant delay in the onset of symptoms. These claims when accepted are allowed on the basis of aggravation of a pre-existing non-compensable condition.

2. Temporary Disability

2.1 When a claim is accepted for a cardiac condition, the injured employee is entitled to full benefits for compensation and medical aid. In most instances, claims are considered on the basis of aggravation of a pre-existing condition, usually atherosclerotic heart disease.

3. Permanent Disability

Permanent disability awards under Section 42 of the Act are granted in cardiac claims where permanent disability results from the injury.

3.1 Permanent disability review is carried out at the point of maximum recovery, usually twelve to eighteen months after the acute occurrence. An assessment is arranged with an outside cardiologist and a report is obtained. The claim is then reviewed and an examination is conducted by the Board's Permanent Disability Medical Specialist in order to evaluate the level of residual disability.

3.2 In the case of traumatic cardiac conditions (cardiac tamponade cardiac contusion, valvular damage, effects of the passage of an electrical current), the total award for Permanent Disability is determined by the Permanent Disability Medical Specialist, is granted to the injured employee and there is no limitation of ongoing entitlement in the claim relative to the compensable cardiac condition.

3.3 Certain limitations apply when determining the Permanent Disability award in claims where allowance has been made on the basis of aggravation of a pre-existing non-compensable condition.

3.3.1 The Permanent Disability award may be reduced in accordance with the Board approved policy on the aggravation of pre-existing conditions and application of the Second Injury and Enhancement Fund (see item 6).

3.3.2 Subsequent to Permanent Disability assessment, ongoing medical treatment is not within entitlement if the need for treatment arises from an underlying vascular disease which pre-existed the accident. An exception to this would be those cases where various surgical procedures, i.e. coronary artery bypass, may be implemented in the acute phase of the disability. Ongoing entitlement may be extended on the advice of the Board's Industrial Medicine Consultant.

4. Subsequent Cardiac Conditions

4.1 When entitlement is established under 1.2.1, 1.2.2, 1.2.3 and 1.2.4, for a cardiac condition, there will be no limitation of ongoing entitlement if the subsequent condition is related to the compensable cardiac condition.

4.2 When entitlement is established under 1.2.5 for a cardiac condition and the condition has stabilized and a Permanent Disability evaluation has been conducted, further entitlement will not be granted for a subsequent cardiac condition unless there is some new industrial occurrence which would merit allowance under a new claim.

5. Fatal Claims

5.1 If death occurs immediately as the result of a compensable cardiac condition, shortly after the initial onset, or while the condition is still in the acute phase, the fatal claim may be accepted and full death benefits may be paid under Section 36 of the Act.

5.2 If death occurs as the result of a new or progressive cardiac condition, the fatal claim is not accepted unless there is a new industrial occurrence which would merit allowance under a new claim.

6. Claims Adjudication Involving Pre-Existing Conditions and Application of the Second Injury and Enhancement Fund

In claims for cardiac conditions, the Board approved policy on the aggravation of pre-existing conditions and application of the Second Injury and Enhancement Fund shall be applied.

7. Claims not Meeting General Criteria

Cardiac claims which do not meet the general criteria shall be individually judged on their own merits. The benefit of doubt applies.

January 21, 1981

CHAPTER V

REFLECTIONS AND RECOMMENDATIONS

1. Reflections on the Compensation of Industrial Diseases

When Ontario's workers' compensation system was first drafted by Chief Justice Sir William Meredith, it was clearly his intention to provide compensation not only for victims of industrial accidents, but also for victims of industrial diseases. In 1913, he wrote:

> ...It would, in my opinion, be a blot on the Act if a workman who suffers from an industrial disease contracted in the course of his employment is not to be entitled to compensation. The risk of contracting disease is inherent in the occupation he follows and he is practically powerless to guard against it. A workman may to some extent guard against accidents, and it would seem not only illogical but unreasonable to compensate him in the one case and to deny him the right to compensation in the other.

What has clearly emerged from this study is that no jurisdiction seems to do well at compensating victims of industrial disease, regardless of whether it uses a schedule of diseases, prefers the use of guidelines or adjudicates each case on its own merit. In fact, Ontario's statistics on compensating industrial disease claims are very impressive in comparison to other jurisdictions. (cf. IB-1 -1.1)

The reasons for the under-recognition of occupational diseases have been addressed at length in this report. Quite apart from conceptual and procedural difficulties in the decision-making process within the compensation system, the lack of adequate training in occupational medicine, the paucity of exposure information and the dearth of research on the multitude of occupational hazards make occupational disease claims very difficult to adjudicate. Although recommendations will be made below to try to minimize these obstacles, it must be appreciated that some difficulties are inherent in the nature of these diseases and will likely persist. The prolonged latency period that characterizes many of the serious industrial diseases, their non-specific nature and the truly multifactorial etiology of these diseases make it essentially impossible to determine with certainty the work-relatedness of any individual claim. At best, it's a question of probabilities.

Epidemiology can indicate that an excess number of cases of a particular disease have occurred as a result of a given exposure. However, it cannot indicate which are the "occupational cases" and which are those which may have occurred even without the exposure. For example, Gibson's foundry study showed that of the 438 foundry workers studied, 8.4 cases of lung cancer were "expected" based on the rate in the general population given the appropriate age structure, however, 18 cases of lung cancer actually occurred. Similarly, in Finkelstein's asbestos study, two lung cancer deaths were expected but 17 were observed. Although the link between foundry exposure, or asbestos, and lung cancer was clearly illustrated by these (and other) studies, there is no way of determining which 8 of the 18 foundry cancers or which two of the 17 asbestos cancers are "non-occupational." In fact, it is not incorrect to surmise that the occupational carcinogen may have played a role in all cases. Equally, it is not incorrect to acknowledge that factors outside the workplace may have influenced the development of the cancer in all cases. (cf. II-A-3.2)

The situation is similar for other diseases. For example, 32.8% of the converter plant workers (smokers), had chronic bronchitis, compared to 9.68% of refinery workers (smokers) who had no sulphur dioxide exposure. The workplace was clearly contributory to the incidence and severity of the disease in the converter plant, yet it would not be scientifically accurate to assume that all cases of chronic bronchitis in this environment are entirely attributable to the workplace. Again, the extent to which the workplace contributed in any individual case cannot be determined. (cf. III-A-3.2.1)

Studies designed to determine dose-response relationships (i.e. the length and/or intensity of exposure required to produce a given disease) are usually fraught with methodological difficulties making them, at best, rough estimates. With respect to cancer there is no demonstrable threshold of exposure below which an increased risk does not occur. Hence, any exposure, however small, may truly be the causative, or at least initiating, factor in a worker's development of cancer. With this in mind, it is not unreasonable to arbitrarily decide that all cancer cases resulting in individuals who have proven occupational exposure to a known carcinogen should be compensable (given a biologically plausible latency period). In fact, this approach has been in effect to some extent in various jurisdictions. (cf. I-B-3)

Still, many cases of occupational cancer would go uncompensated, as proof of exposure and solid evidence of carcinogenicity are scarce. While considerable over-compensation would occur in some situations, vast under-compensation would still be a reality. Unfortunately, to compensate cancer in anyone who has ever been exposed to a possible carcinogen in his or her workplace irrespective of length or intensity of exposure may well amount to compensating almost every cancer case in anyone who ever worked.

Similarly, to compensate respiratory disease in anyone who was ever exposed to proven respiratory hazards, or any cardiac death in anyone who has ever worked in a work environment known to be associated with excessive stress, or any heavy labourer who develops degenerative disc disease, and so on would, compensate some cases where in fact non-workplace factors may have been predominant, while vastly undercompensating many others exposed to potential but as yet unstudied hazards.

Occupational factors may effect every aspect of a worker's health (cf. I-A-2); the scope of occupational diseases is such that research will never be able to completely catch up with all possible hazards. The extent of the shortfall in compensating occupational diseases depends largely on how "occupational" and "disease" are defined. (cf. I-A-3) It must be concluded that the only way to ensure the compensation of all disabled workers for whom working conditions may have been contributory to their condition is essentially to compensate all workers. If those indirectly effected by industrial hazards are also to be included (such as the families of asbestos workers exposed to fibers brought home on work clothes, or those living downwind from coke ovens) then the system may truly have to compensate everyone.

The comprehensive system implied by this conclusion has met with considerable opposition. First, there has been concern from labour and injured workers' organizations that in the present political-economic conjuncture, workers would receive inadequate benefits if the system was broadened. From the employers' point of view, the provision of adequate benefits to all would be unacceptable both economically and philosophically. Concerns have also been raised regarding the administration of such a system. Secondly, although the apportionment of cost would no longer be of direct concern to the claimant, the problem of how much of the bill should be paid by industry would have to be faced. Labour organizations have pointed out that much of the research in occupational disease done in this province has originated directly from compensation battles and hence they are concerned that a comprehensive system would dull the incentive for such needed research. Thirdly, and directly related to the previous point, concern has been expressed that the incentive to clean up the workplace would be lessened.

Although a full discussion of the relative merits of these arguments is beyond the scope of this report, it is worth noting that aside from the inherent desirability of providing an adequate standard of living to all victims of disease and their survivors, it would seem that the ultimate success of such a system would largely be determined by the extent to which its funding could eventually become truly causation-based. For example, if 80% of lung cancer is attributable to cigarette smoking and 20% to occupational exposures (the one-cause/one-effect fallacy notwithstanding), the full cost of disability due to lung cancer should be borne by taxes on cigarettes and corporate taxation. The latter would be divided among the asbestos industry, steel industry, uranium industry, nickel industry, etc. according to the best available knowledge as to their relative contributions.

To throw up our hands in frustration stating that the vast majority of causes are not known and therefore funding must be borne non-specifically by society at large, minimizes the potentially enormous impact this system could eventually have on prevention (in conjunction with enforced standards and other public health measures). To the extent that society truly does not know the cause of disease, research funding must be allocated to find out. (With respect to cancer, a large case-control study, based on exposure histories may be a starting point-- cf. II-A-5.) In this way, generations to come will better apportion the cost of disability. In turn, the extent (and cost) of disability would be increasingly reduced as the effect of this cost would be expected to result in preventive measures and improved health practices. The larger the "unknown" factor, the less effective the system.

Many other issues would also have to be addressed before the desirability of such a scheme could be properly considered. For example, while a comprehensive system would clearly remove the burden of determining the work-relatedness of each claim, it would not necessarily remove the problems of assessing impairment and disability. Many of the cases presented above illustrated dissatisfaction not with respect to the Board's decision regarding work-relatedness of the disease, but with the permanent partial disability rating. In fact, Barth noted that "no other issue has so consistently stirred controversy regarding workers' compensation for asbestos-related disease as has the basis for rating permanent partial disability benefits."

It is usually assumed that those workers who are totally disabled (or too ill to work) would be covered by a comprehensive system based on lost wages. However, it would have to be decided how to compensate those workers still able to work but who endure pain, suffering and diminished quality of life as well as reduced potential earning capacity as a result of an impairment. One possibility is to include only those totally disabled in the comprehensive system, and to continue to provide an impairment-based lump sum or permanent partial disability award through the workers' compensation system to those who are still working but who have suffered a work-related impairment (e.g. noise-induced hearing loss, vibration-induced white finger, etc.). This would imply that those people with non-work-related partial disabilities would be neither eligible for workers' compensation benefits nor for comprehensive disability benefits. Attempts to establish the work-relatedness of a disability would therefore continue on a case-by-case-basis with the most seriously ill, the total disabled, not required to pursue the issue of cause.

Clearly the other possibility is to base the comprehensive system not only on wage-loss but also on impairment, with entitlement to partial disability awards available to all regardless of the work-relatedness of the source of pain or suffering. Regardless of the eventual system used to determine work-relatedness, the issue of disability assessment must be addressed.

It was found that currently the vast majority of industrial disease cases compensated are for short-term disability. It would be possible to maintain workers' compensation for short-term disability, less than 3 or 6 months, for example. This option, the inverse of the Swedish model, would be desirable from the point of view of maintaining (almost) full income for those temporarily unable to work as a result of a work-related condition, but has the drawback that it would not eliminate the battles needed to establish that the individual case of disease is caused by an occupational exposure (unless presumptions are established through a schedule.)

Since considerable attention would be required to find methods of resolving these issues, interim measures could be adopted to rectify some of the problems in the present system. Recommendations with respect to both the decisions regarding work-relatedness and the decisions regarding disability assessment by the Workers' Compensation Board will be presented in Section 3 below.

2. Recommendations About Matters External to the WCB

It has become clear that while the rejection rate of industrial disease claims is unacceptably high, the under-reporting of occupational diseases is an even larger factor in the undercompensation of industrial diseases. (cf. I-B-1, II-B-2, III-B-1) Reform in workers' compensation policy and the process of adjudication may in itself stimulate the filing of more claims. However, various measures outside the domain of the WCB are also clearly necessary if a greater number of workers suffering from occupational diseases are to be identified and appropriately compensated. Thus recommendations on matters not strictly within the jurisdiction of the WCB will be presented first.

2.1 On the State of Medical Knowledge and the Organization of Occupational Medicine Services

Many work-related diseases are not likely to be diagnosed as such, simply because no one has taken an occupational history. Physicians in general lack awareness of occupational hazards as they are not well-trained in this area. (cf. I-A-3.3) The need to upgrade the occupational health content of medical school curricula has been increasingly recognized, and plans are currently underway in at least one medical school in this province to integrate occupational medicine thoroughly into its undergraduate program. Other schools should be actively encouraged to follow this example.

While some occupational health training is provided in residency programs for specialists in community medicine and seminars on occupational health are beginning to be included in some family medicine residency programs, there is growing consensus that this is still not adequate.

The Ministry of Labour and the Workers' Compensation Board should also be prompted to use their influence to lobby the Royal College of Physicians and Surgeons to elevate occupational medicine to the specialty which it is. Further, adequate funding for postgraduate and residency programs in this field must be ensured.

The WCB and OHIP may also consider amending their payment schedules to specifically recognize the value of occupational history-taking by providing premiums to physicans who take the time to educate patients about preventive techniques and about the signs, symptoms and mechanics of diseases which they may be in danger of contracting. In this regard, the provision of preferential billing rates to physicians who maintain accreditation in occupational medicine should also be considered.

A further issue alluded to by some practitioners as a factor in the failure of physicians to file workers' compensation practitioners claims, is that the WCB does not pay the physician's fee until the claim is adjudicated. A simple check-off on OHIP billing cards as to suspected work-relatedness would not only circumvent this problem but would facilitate better record-keeping on occupational diseases. Transfer payments from the WCB to OHIP can then take place when the adjudication has been completed so that industry would bear its fair share of health care costs.

As the reality of labour-management conflict in the occupational health arena cannot be ignored, the concentration of occupational medicine expertise in management's jurisdiction is potentially problematic (cf. I-A-3.3). It would be worthwhile for the Ministry of Labour and the WCB to ensure that labour organizations as well as corporate enterprises have full access to such expertise. The Canadian Centre for Occupational Health and Safety provides an information service to some extent, and the Advisory Council on Occupational Health and Safety has recommended that more funding be allotted to the educational program of the Ontario Federation of Labour, and other labour projects. However, the individual worker must be assured of access to expert advice concerning his or her own medical problems in an environment which is completely free from possible outside pressure and which would inspire the trust that medical confidentiality is completely respected.

In this regard, independent occupational health clinics should be encouraged and workers must play a key role in the selection of the medical staff who are to service them. In fact, a system of delivering occupational health services based in these units, merits careful consideration. The elevation of occupational medicine to a specialty may itself encourage the growth of university-affiliated occupational health clinics. In any case, the greater the scientific expertise in the area, the less the likelihood that political considerations will be allowed to intervene. Moreover, by raising the level of knowledge in the medical community regarding occupational diseases and ensuring unhindered access to this expertise, not only would more cases of industrial disease be properly recognized, but much of the frustration stemming from conflicting advice from ill-informed but well-meaning practitioners (cf. III-B-2, etc.) would undoubtedly be reduced.

2.2 *The Maintenance of Exposure Records*

Another problem encountered in many of the cases reviewed was related to the difficulty in ascertaining to what substances and to what degree a worker has been exposed in his present or previous employment. It has been suggested that the under-reporting of occupational diseases is largely attributable to the absence of records needed to alert workers and their physicians to the possible role of workplace factors in their diseases.

As discussed (cf. II-A-4.2), Ontario's designated substance Regulations, if they are followed, should aid considerably. However, the scope of occupational hazards far exceeds the few substances proposed for regulation in the immediate future.

A few of the large companies in the province have taken steps to compile personal exposure records. Certainly other measures could be taken by the Ministry of Labour without prohibitive effort and cost to ensure widespread record keeping. For example, notification of all new substances could be provided not only to the Director, as indicated by the Occupational Health & Safety Act, but also to each worker and possibly to his or her physician. Also, joint health and safety committees could be encouraged not only to design workplace monitoring and control programs, but also to generate a system of personal exposure records. Industrial hygiene/occupational health departments, where they do exist, should be directly responsible to the joint committees so that the latter could more effectively perform their function.

In principle, records should be kept on all exposures referred to in the American Conference of Governmental Industrial Hygienists (ACGIH) guidelines as a minimum. Although the list contains some 400 hazards, only a few are likely to be pertinent to each plant. Efforts are apparently underway at the Ministry of Labour's occupational health branch to computerize exposure information obtained on workplace inspections. This could facilitate the future adjudication of industrial disease cases arising from these environments in the event that control measures fail to prevent untoward effects.

The practice of filing WCB claims at the time excessive exposure, especially to a carcinogen, is known to have occurred, should not be discouraged. (Claims of this nature constituted 14% of cancer claims in the sample reviewed.) However, it may be advisable to develop a separate coding system clearly delineating these files from those in which a disease has actually occurred.

The division of responsibility between the Ministry of Labour and the WCB with respect to exposure records merits careful consideration. Suffice it to emphasize that the development of an efficient system of recording exposure information is imperative to the swift and just adjudication of workers' compensation claims.

2.3 Responsibility for Research

This study has shown that claims for potentially work-related diseases are almost systematically rejected in the absence of strong supportive research. Animal studies or short-term carcinogenicity tests are important in decisions to regulate a substance as a carcinogen, and should certainly influence a company's decision whether or not to use or manufacture a given substance. However, to date, evidence of this nature has played little role in the adjudication of claims (for reasons discussed in II-A-3.1).

Epidemiological evidence has been (and probably will continue to be) the key to the recognition of diseases as work-related. However, even epidemiological evidence is often considered unsatisfactory for adjudication purposes in Ontario. For example, if the research was carried out in other parts of the world where working conditions were not necessarily parallel to those existing in Ontario. In fact, industrial processes and working conditions may differ substantially from one plant to another even within the province, accounting for the reluctance on the part of the WCB to generalize findings from one plant to others (e.g. cancer in foundries, cf. II-B-3.2; cancer in nickel refinery workers, cf. II-B-3.3.)

The need for greater resources to be allocated to research into industrial disease is thus obvious from the compensation point of view. Epidemiological studies, although not to be used as substitutes for a cautious approach in the regulation of industrial hygiene do indeed have a role to play in prevention.

One possible solution is to create a single research agency, council, or branch entrusted with the responsibility of conducting needed studies in industrial disease both for purposes of compensation and prevention. WCB funded accident prevention associations could be encouraged to allocate some of their resources to a separate industrial disease research agency. The Ministry of Labour's special studies branch could be incorporated into this body. The agency could decide to contract University-based groups to perform some of its studies.

This agency or council could play a major role in disseminating information on occupational health matters, and at a minimum, should be required to publish its studies. Scientists have a very important role to play with respect to assessing protocols (i.e. the validity of the methodology proposed to investigate a problem), but determining funding priorities is not strictly a scientific matter. It is imperative that the research council be responsive to the needs of working people. Moreover, this council must be made accountable for its decisions, and therefore it would seem appropriate for it to be placed within the jurisdiction of the Ministry of Labour.

2.4 Data Collection and Occupational Disease Registries

Both for purposes of adjudicating industrial disease claims, and for research purposes, it is necessary to improve the state of data collection. Efforts to accomplish this were reviewed elsewhere in this report (cf. II-A-4.2). Suffice it to state that the avenues currently being pursued show great potential and are worth encouraging.

The establishment of a system of independent occupational health clinics would also undoubtedly facilitate the recognition and reporting of occupational diseases.

The WCB certainly also has a strong role to play in the identification and reporting of occupational diseases. It could use this influence, for example, to assure that cancer treatment centers record appropriate data about occupational factors in cases and to encourage the performing of autopsies in all deaths from potentially work-related diseases.

The WCB houses a great deal of very rich data regarding occupational diseases. At present scattered throughout the different divisions and branches of the Board, these data should be systematically gathered and computerized for study purposes. At a minimum, records should be maintained on rejected claims as well as those allowed, with coding for diagnosis, suspected exposure and workplace. Moreover, the Board should be urged to allow its data to be used by bona fide researchers for studies that might lead to the prevention of occupational diseases and to better medical management of victims of these conditions. Strict observance of the ethics of consent and confidentiality must, of course be enforced with respect to these data. The Board should also be encouraged to conduct its own studies, such as suggested by Barth, to determine the accuracy of its medical assessments by comparing them with autopsy findings.

2.5 *Outreach*

Even if medical knowledge on occupational diseases was at a much higher level than it is today, and even if exposure information was available, the research was done and the data were collected, there is no assurance that all appropriate claims would be filed. Thus another function external to the actual policy making and claims adjudication process of the Board is to ensure that workers, their representatives, their physicians and all other concerned parties are properly informed of WCB policy and procedure.

The lack of knowledge regarding rights to compensation has been alluded to. Although as Barth has described, the Board has made commendable efforts to alleviate this lack of information, there is room for improvement. As a first step, consideration should be given to expanding the current occupational disease pamphlet produced by the WCB, to provide more detailed information on claims procedure, and to include the schedule of diseases and all formulated guidelines. A wide distribution of this pamphlet could not only bring in more worthy claims, but would eliminate some of the frustration that stems from lack of understanding about the Board's approach to adjudicating occupational disease claims.

Much use is currently made of the media to encourage accident prevention (e.g. TV advertisements). The media could also be used to promote prevention of occupational disease, and to educate workers about their rights to compensation. Advertisements in public transit may also be effective.

3. Recommendations Regarding WCB Policy and Procedure

Once the appropriate claims are submitted to the Board, they must be promptly and fairly adjudicated . Some recommendations aimed at improving this function are presented below.

3.1 Policy Formulation

3.1.1 On the Work-Relatedness of Disease

The approach to deciding the work-relatedness of a given disease differs considerably from jurisdiction to jurisdiction (cf. I-B-3). It was noted that in Ontario although the schedule of diseases has not been expanded, a large variety of diseases have been compensated. It was also noted that some diseases are compensated even if they do not meet the "peculiar to or characteristic of" requirement. Nevertheless, an extensive review of cancer and chronic respiratory disease claims clearly illustrated that claims of this nature which are addressed neither by guidelines nor by the schedule of diseases had a very slim chance of being allowed.

In 1967, in his Report on the compensation system in Ontario, Justice McGillvary wrote:

> If I felt from the evidence...that any workman is likely to suffer from a failure to list a disease...I would have no hesitancy in recommending that the Schedule be amended to list all such diseases no matter how extensive the list would be or how often it would need to be amended. Suffice it to say that I am not so convinced, particularly so when I note that barely 4% of all claims submitted to the Board are turned down. As a consequence, I shall make no recommendations in this Report for any addition to Schedule 3.

As shown in this study, the rejection rate for serious occupational diseases is much higher than McGillvary thought. In fact, well over half the cancer and chronic respiratory disease claims filed were rejected. As adjudicating each case on its own merit is not only time-consuming but also results in a great number of rejected claims, consideration should again be given to expanding the schedule.

A distinct advantage to the ongoing development of a schedule of occupational diseases is that it would tend to focus the debate less on individual cases and more on the content of the schedule, thereby removing some of the pressure from the individual claimant to prove his or her case each time.

Just as it was found that the vast majority of claims which do not meet guidelines are rejected, all but one claim in the survey which did meet the guidelines was allowed. In practice, the guidelines seem to carry the same presumptive weight as the schedule of diseases. The major difference lies in the many restrictive clauses contained in the guidelines.

The underlying methodological difficulties (cf. II-A, III-A) and the scientific background on which the guidelines are based (cf. II-B, III-B) were extensively reviewed. Although Ontario's guidelines are not necessarily more strict than other jurisdictions (cf. II-B-1), it must be acknowledged that the rationale for the actual numbers chosen or even the specific parameters included is often tenuous. Moreover, the absence of widespread input or even public awareness of the formulation of guidelines (in contrast, for example, to the procedure for setting standards for the designated substance Regulations), is clearly a source of suspicion and distrust. To quote Barth: "It is difficult to understand how an agency that has lived so much under public scrutiny has avoided becoming more open than it has in the matter of these guidelines in so controversial a field as industrial disease."

It would seem that the in-house procedure as well as the rather conservative approach in the formulation of guidelines relates at least in part to the strong role played by the Board's medical staff.

Notwithstanding the fact that there are definite scientific guidelines to help decide which studies can be considered relatively sound and which can be largely discounted, review of the Board's practice suggests that the Board doctors have a strong tendency to discount less convincing data that would militate in favour of broadening the allowance of claims, while they tend to exalt weak studies that "support" the lack of a relationship between a disease and an occupational exposure. In other words, in the absence of strong positive evidence, the assumption of no increased risk prevails.

This may relate at least partially to the inherent approach to causation that exists among scientists. As many scientists seem to feel that their prime responsibility is to avoid misleading the profession and causing panic, they generally tend to be cautious about accepting new theories or evidence as valid. When confronted with a great deal of uncertainty, scientists usually prefer to avoid the stigma of being wrong by deferring judgement and instead calling for additional study and research. Some people feel, however, that the Ontario WCB doctors are particularly conservative in their approach. This is thought to be based upon their concern that if the criteria for allowing claims are liberalized, this will open the flood gates and bankrupt the province's industries. (In light of the huge sums of money spent on investigating claims, tracking down exposure information and conducting study after study to delineate the restrictions, one wonders if the straightforward allowance of more claims would in fact be more "cost-effective" anyhow. A cost analysis of this hypothesis might be quite appropriate.)

In any case, although an understanding of scientific knowledge on the various issues must be a starting point for decisions about the work-relatedness of disease, it is well-recognized that humanitarian, administrative, economic and political considerations all come into play in the formulation of any policy. In fact, the very definition of "occupational disease" is not strictly a scientific question. It is therefore important that the decision-making structure reflect this reality and cease to mystify policy in the name of science.

It was recommended above that the explicit responsibility for evaluating literature and carrying out the necessary research to provide insight into the work-relatedness of disease should be allocated to a separate agency or council, responsible for publicizing its methodology and findings. Based on the information accumulated, a detailed schedule of diseases could be developed and frequently updated. More liberal use of research from other jurisdictions and especially from international agencies should also be encouraged. For example, a table adapted from the IARC listings could serve as a starting point for compensating occupational cancer, with definitions of exposure circumstances consistent with the most broad interpretation of the international body of knowledge and utilizing minimum biologically plausible latency periods. Submissions from all interested parties should be requested and public hearings held periodically to amend the schedule as new diseases and hazardous processes are defined. The responsibility for establishing the schedule should rest with this council.

Cases which do not meet the conditions outlined in the schedule should also be carefully considered by the WCB and it would be anticipated that a fair number of these should also be allowed on their own merit.

3.1.2 On Disability Assessments and Pre-Existing Conditions

Even when a disease is clearly designated as occupational, silicosis for example, problems arise due to the conceptual confusion about what exactly is being compensated.

The "dual" approach to compensation benefits, which Paul Weiler advocated in a 1980 report about reforming workers' compensation, involved compensating for impairment (either by a lump sum or periodic payments) as well as for disability, which he defines in terms of wage loss. The Weiler approach would distinguish the calculation of physical impairment from the assessment of disability. The fact that disability factors must come into play in assessing impairment in the current WCB benefit scheme, has created confusion (cf. III-A-4) and may, in fact, continue to pose problems. The cases presented above are testimony to the frequent hesitancy of the Board doctors to broaden their impairment assessments from those strictly measurable by clinical techniques. Many people are therefore withholding judgement on the dual benefit structure as well as the "deemed" ability to work clause in the Government's 1981 White Paper due to concerns regarding the implementation of this policy.

Also a source of controversy is the Board's practice of trying to isolate the impairment attributable to a compensable disease from that due to other medical conditions either of the same organ system (e.g. trying to isolate the impairment due to silicosis from that due to chronic obstructive lung disease) or of different organ systems (e.g. stating that a worker's disability is due to his arthritis not his respiratory condition). Quite apart from the fact that these distinctions are often difficult to clinically determine with a high degree of precision, it is also inconsistent with the Board's general approach to providing compensation for the aggravation of pre-existing conditions and the philosophy of "taking a worker as he or she is found".

It is therefore recommended that when an occupational disease contributes significantly to the disability, or for that matter, death of an individual, the total disability or fatality should be deemed compensable. This would mean that if an occupational disease significantly contributes to a worker's inability to continue at his or her job, full wage-loss benefits would be paid, regardless of the presence of other factors. For example, a silicotic with arthritis and diabetes who is unable to work would be entitled to full wage-loss benefits as long as the silicosis accounted for a significant portion of this disability.

Clearly, debate would focus on the definition of "significant". As was argued with respect to the advantages of a schedule to guide decisions about the work-relatedness of a disease, it would spare the claimant undue suffering if a detailed schedule could be developed to define how the impairment is to be measured and the level of impairment necessary to automatically qualify (e.g. X% reduction in FEV_1). The process of developing this schedule would be considerably controversial (cf. III-B-1.2) and, as was recommended above, should have input from the public at large as well as from scientists. Although cases which did not meet the minimum specifications decided upon would still be assessed on their own merit, with factors such as age, rehabilitation potential, etc. taken into consideration, the existence of this guide could considerably diminish the delays and inconsistencies that characterize the present method of case by case assessment.

Also, lump sums or other forms of permanent partial disability awards should be based on the full extent of impairment of at least the organ system involved. The attempt, for example, to distinguish smoking effect from dust effect is not only clinically difficult but is also contrary to the principles of workers' compensation.

As controversy would also still exist over whether a compensable disease, for example, asbestosis, played any role at all in a death, for example, myocardical infarction, it is recommended that the causes of death to be deemed compensable in association with a specific occupational disease, also be carefully defined in the schedule of diseases. Again, cases which do not meet the requirements should still be adjudicated on their own merit.

If P.D. benefit reforms were to replace the present system which pays impairment-based pensions, with benefits taking the worker's age and rehabilitation potential into account, the decision about eligibility for rehabilitation as described above should no longer discriminate against older workers or those less suitable for the job market. These workers would also probably be eligible for wage-loss benefits.

(Although the details of cost apportionment are beyond the scope of this report, a portion of the cost should probably be allocated to a special fund established for the purpose of compensating industrial diseases of multiple causation.)

3.2 Issues Pertaining to Claims Procedure

The solution to many of the problems identified in claims procedure would follow directly from the above recommendations. For example, the role of the Board's medical staff, the adjudicator and the Claims Review Branch would be guided to a much greater extent by the Schedule of Diseases, with the existence of outside appeal tribunals and medical review panels, as suggested by the White Paper, to serve as safeguards.

As permanent disability assessments would take into consideration the entire extent of disability, not merely the impairment directly attributable to the specific disease, the contentious issues raised, for example, with respect to the Advisory Committee on Occupational Chest Diseases would be less pronounced. Nonetheless, the practice of having a Board doctor screening out claims must be looked at more closely and certainly a much better explanation must be given as to the reasons behind Board decisions at every level, regardless of the method used to assess disability. The ACOCD must function in a formal manner keeping minutes of meetings, summaries of which would be provided in the claim file.

The initial decision with respect to the advisability or the ability of the claimant to continue at work could rest with the claimant's own medical practitioner. If the Board's medical staff disagrees with this assessment, the claim could immediately be referred to the Appeals Tribunal which would receive aid from the Medical Review Panel. This should considerably reduce the delay in deciding appeal cases. The Board should also be much freer in authorizing wage-loss benefits while the claim is being investigated (cf. III-B-3.1). Those workers temporarily unable or ill-advised to remain at their jobs due to occupational hazards, e.g. a worker with a high blood lead level, must be assured of wage-loss benefits even if no clinical impairment is demonstrable. (In this regard, it is interesting to note that Quebec's Occupational Health and Safety Act provides full compensation to a pregnant woman who is advised to leave her job to prevent possible ill-effects to her fetus.)

Finally, the assessment of the workplace must take on greater importance in claims adjudication than is currently the practice. Although there is some merit in the task being carried out by the Ministry of Labour so as to avoid duplication, the resources available to the Ministry do not seem adequate to even handle the immense tasks that it presently faces. Accordingly the Ministry's resources must be greatly expanded and/or the WCB should establish a team of industrial hygienists to work in conjunction with the medical staff. Use of industrial hygienists should not be subject to the opinion of the medical staff, but to the adjudicator; and the status of the industrial hygiene report should be equivalent, not subordinate to, medical opinion. In this regard it may be useful to expand the Medical Review Panel to an Industrial Hygiene--Medical Review Panel, or to create a separate review panel to offer opinions on workplace conditions if necessary.

4. Summary

Although no system of workers' compensation could be expected to fully compensate workers who suffer from work-related disability, the following recommendations may be of value:

A. Matters External to the WCB

1. Re: Medical Community

   (a) Medical schools should be encouraged to increase the occupational health content of their curricula.

   (b) The Royal College of Physicians and Surgeons should be encouraged to designate occupational medicine as a specialty.

   (c) Funding for postgraduate and residency programs in occupational medicine should be provided.

   (d) Physician payment schemes should reflect the value of occupational history taking and the maintenance of accreditation in occupational medicine.

   (e) Consideration should be given to providing a check-off system on OHIP billing cards to indicate work-relatedness.

   (f) Independent occupational health clinics should be encouraged and steps should be taken to explore the possibilities of developing a system along these lines.

2. Re: Exposure Records

   (a) Notification of all new substances to be introduced into the workplace should be provided to each worker and the appropriate physician.

   (b) Joint health and safety committees should generate a system of personal exposure records appropriate to their workplace. (Inclusion of all ACGIH-assessed hazards should be considered the minimal requirement.)

   (c) The filing of WCB claims at the time of suspected excessive exposure is not to be discouraged at least until an alternate system of documenting exposure is fully in place.

3. Re: Research

   A specific research agency, council or branch, should be established which would be responsible for conducting needed studies in industrial disease both for purposes of compensation and prevention. This body must be responsive to the needs of working people and funding priorities should be decided with the input of non-scientists as well as scientists.

4. Re: Data Collection

   (a) The WCB should use its influence to assure that data on occupational factors are recorded at cancer treatment centers.

   (b) Autopsies on all possibly work-related deaths should be encouraged.

   (c) Records of all claims filed at the WCB should be coded for diagnosis, suspected exposure, workplace and ultimate claim status.

   (d) Board data should be made available to bona fide researchers to conduct studies towards the prevention of occupational diseases and improved management of victims of these conditions.

5. Re: Outreach

   (a) The Board's Occupational Disease pamphlet should be expanded to include more detail on Board policy, and widely distributed.

   (b) The media and public transit could be used to promote the prevention of occupational disease as well as accidents, and to inform workers of their right to compensation.

B. Board Policy and Procedure

6. Re: Work-Relatedness of Disease

   (a) Data obtained from the proposed research agency as well as from the IARC and other jurisdictions should be used to formulate a detailed schedule of occupational diseases.

   (b) Submissions from all interested parties should be requested and public hearings held to facilitate the periodic amending of the schedule.

   (c) Claims which do not meet the conditions described in the schedule should be adjudicated on their own merit.

7. Re: Disability Assessments and Pre-Existing Conditions

   (a) Disability assessments should reflect the total extent of impairment both directly and indirectly related to the compensable disease.

   (b) Wage-loss benefits should be paid to any worker who either cannot continue working or who should not because of the combined impact of an occupational disease and a non-occupational condition.

   (c) The definition of "significant" impairment should be specified in a schedule; those not meeting these minimum requirements should be assessed on their own merits with regard to factors such as age and retraining potential taken into consideration.

   (d) The causes of death to be deemed compensable in association with various occupational diseases could be defined in the schedule of diseases; those not meeting these conditions should be adjudicated on their own merit.

8. Re: Claims Procedure

   (a) Detailed explanations for Board decisions should be provided to the claimant at each level of decision-making.

   (b) The initial assessment of the claimant's ability to work should be made by his or her physician. If the Board disagrees, the claim should be directly forwarded to the appeals tribunal and medical review panel.

   (c) The provision of wage-loss benefits during the investigation of a claim should be more liberally authorized.

   (d) Industrial hygiene assessments must take on greater importance. Consideration should be given to expanding the medical review panel to include industrial hygienists, or creating a separate review panel of industrial hygienists.